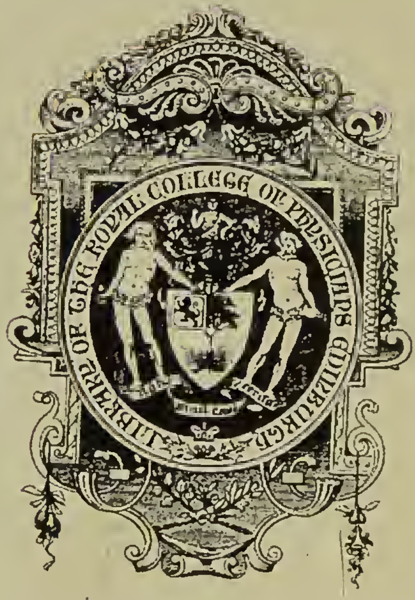

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VOLUME CXLVII.

PREFACE.

The following papers selected from Sir W. Gull's published writings have been reprinted with only such corrections as were absolutely necessary. They have been classified according to the pathological views expressed by the writer, so that the paper on "A Cretinoid State" (p. 315) will be found under Diseases of the Nervous System, and that on "Chronic Bright's Disease" (p. 375) under Diseases of the Vascular System. Under the several headings the various articles have been arranged in chronological order, so that the progressive views of the writer may be traced by reading them consecutively.

The paper on the Effect of Ether on Various Classes of Animals (p. 571) is incomplete, but its historical interest is so great that it could not rightly have been omitted. No record of the original (even if it was ever written) has been found. No apology is needed for reprinting the letter on Mr. Cock's case (p. 575). It carries the reader back to the days when an operation under the influence of chloroform was of such rare occurrence as to be thought worthy of special note.

It is hoped that no paper of importance has been omitted from the present volume, but it has been difficult to trace writings scattered through current medical literature over a period of more than forty years, since no list of them had been kept.

Some of the papers which have been reprinted were originally published in collaboration with other writers, and this has in each instance been duly acknowledged.

The majority of Sir William Gull's lectures on Comparative Anatomy, Physiology, and Clinical Medicine were delivered from notes only, which accounts for the fact that there is no written record of that part of his teaching which occupied so large a portion of his early life.

A Report on the Morbid Anatomy, Pathology, and Treatment of Epidemic Cholera has not been reprinted, as it is included in the Reports on Epidemic Cholera issued by the Royal College of Physicians.

The Addresses will be issued in a subsequent volume together with a Biographical Memoir.

Some difficulty has been encountered in reproducing the plates, especially those illustrating the paper "On Changes in the Spinal Cord and its Vessels in Arterio-Capillary Fibrosis." The original drawings had been lost, but were discovered in time to correct the proofs of the reproductions. Owing to the efforts of Mr. Newman a satisfactory result has been obtained.

Thanks are due to Mr. Coldrey, late Assistant Librarian to the Royal Medical and Chirurgical Society for much valuable help, to the editors of various publications for permission to reprint the several papers, and to those whose liberality has made it possible to issue the present volume in a form which it is hoped is not unworthy of the great physician and teacher whose life's work it represents.

T. D. ACLAND.

March 14*th*, 1894.

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SECTION I.

DISEASES OF THE NERVOUS SYSTEM.

(*a*) DISEASES OF THE BRAIN.

ON ABSCESS OF THE BRAIN.[1]

The brain exhibits in disease a tendency to suppuration which gives it a pathological rank with glandular organs. The bearing of this upon the therapeutics of cerebral affections in general, is both obvious and important, especially as to the use of mercury, since it is admitted that the diseases of tissues having this predisposition, do not bear the full action of that remedy.

A recent excellent writer[2] admits the occurrence of idiopathic cerebral abscess. I suppose he means no more than abscess whose origin is unaccounted for, since we have no evidence of any such intrinsic perversion of the nutrition of the brain as leads to suppuration. The nearest approach to such a result is in scrofulosis, but then only where the scrofulous deposit acts as an extraneous substance upon the tissue in the same manner as an hydatid cyst; in neither of which cases can the suppuration be considered idiopathic.[3] A perusal of the cases given by Abercrombie may have favored the opinion referred to, as he seems tacitly to assume an independent origin in many of them. But it is to be remembered, in contradiction to such an inference, that the cases he records are principally intended to estabish the fact of suppuration, the different forms of it and its general symptoms, and not avowedly to trace the causes. This of necessity is a later subject of inquiry.

It is in, what has been called, "metastatic abscess," that the causes are apt to be overlooked. The suppuration in the brain often appears as an isolated affection, and the local disease which gives rise to it is regarded as a mere coincidence. It is, indeed, only by multiplied examples that we can get to see the relation between the primary disease and the secondary effects. For instance, Abercrombie records the case of a gentleman who for years had been subject to cough and purulent expectoration, with cirrhosis of the lung, and who died of secondary abscesses in the brain. The connection between the disease in the chest and in the brain would, from this one case, appear only as a coincidence, but taken with Cases 12, 13, 14, (36, 37, 38)[1] given below, we should be prepared at the bed-side for a recurrence of the phenomena. These cases of secondary abscess are amongst the most obscure, whether we regard their insidious origin and latent course, or the primary diseases which give rise to them. The brain is especially liable thus to suffer, and in instances where the liver and lungs are unaffected. It was so in Case 9 (33), where ileitis set up abscess of a mesenteric gland, which in its turn was the cause of many secondary abscesses in the brain, and in Case 8 (32) chronic abscesses of the abdominal wall was followed by several abscesses in the brain, though the other viscera escaped. Case 9 (33) is the more striking since, according to our present theories of purulent infection, the capillaries of the liver ought to have suffered first. When the primary abscess is at a distance, the blood appears to be the only medium through which the morbid influence can be conveyed, but the steps of the process are not yet clear.

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[1] Reprinted from the 'Guy's Hospital Reports,' vol. iii, 1857, p. 261.

[2] Lebert, "Über Gehirnabscesse," 'Archiv für pathologische Anatomie,' &c., Bd. x, 1856.

[3] The distinction here insisted upon is obviously more than a verbal refinement. Daily experience proves how much clinical investigation is prevented by the inappropriate use of the word idiopathic.

Although we have used the word *"metastatic,"* we cannot admit the validity of the theory in the present subject. We have no evidence that a distant tissue begins to suppurate by some mysterious transference of force, like the sinking of one end of a balance when the weight is lessened at the other. Something of this kind may occur in the natural functions and growth of parts, but it is probably limited to the processes of nutrition under the regulation of the reflex working of the sympathetic system, and does not extend to such wide perversions as the formation of pus. The theory of the transmission of the morbid action through the cerebro-spinal nerves which some have endeavoured to establish by such instances, as the removal of a testis, followed by abscess in the opposite lobe of the cerebellum, is equally untenable, these cases being in all probability examples of secondary abscess through the blood, the seat of which is accidental.

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[1] The numbers in brackets throughout this paper refer to a table of symptoms and causes of abscess of the brain reprinted from 'Reynold's System of Medicine,' *see* pp. 65—84. ED.

With this tendency to secondary suppuration in the brain is also to be noted the fact of its latency. A person may perform all his duties and be in apparently good health, though for many months he may have a large abscess in the cerebrum. Case 13 (37) is a remarkable illustration of this. The patient was even amused by the involuntary jerkings of the arm which ushered in the fatal inflammation around the cyst.

The latency of cerebral abscess seems explicable by the combination of several facts. It is generally seated in the substance of the hemispheres, where it is known that extensive disorganisation may go on without any indication, provided the corpora striata, thalami optici, and other central parts be not involved; the cerebrum and cerebellum appearing to have, like other organs, a surplusage not required on ordinary occasions.

Encysted abscess probably forms slowly, and does not so much destroy as compress parts, and when the compression is gradual and uniform there is a yielding which, up to a certain point, is compatible with function.

In many instances even at last it is not the abscess itself which occasions the symptoms, but the reactive inflammation around it.

The course of cerebral abscess is in our present experience always a fatal one. There are no known cases where the sac has contracted, and the pus dried up, as occasionally happens with chronic abscess in other parts. "At first sight," says Lebert, "it may appear remarkable that, with so frequent encysting of the abscess, and with a latent course, often of many months, no instance of real cure should be known, whilst in softening and effusions of blood the formation of a cyst is the first step in the healing process. The cyst which surrounds the abscess is, however, altogether different from those mentioned. It is thicker, more vascular, and partakes of the quality which pyogenic membranes have of pouring out pus, so that instead of being absorbed, the quantity is increased, the pressure on the brain heightened, and the way prepared for an unfavorable course as well as a fatal termination."

We can hardly admit that the structure of the cyst is alone the cause of the unfavorable issue, since there is nothing in it different from that of chronic abscesses in general, which occasionally do contract and dry up. In Case 14 (38) the pus was actually beginning to undergo the earthy infiltration indicative of such a process. It is probably rather to other circumstances than this that we must refer the fatal result. The pus of cerebral abscess is prone to undergo decomposition. All observers have noticed the occasional fetor of these collections, quite apart from any communication with bone or the external air. In this occurrence, and in the extreme alkalinity and mucoid character of the pus, which is so frequent in these cases, we cannot but recognise chemical peculiarities which oppose that quiescence of capillary action necessary to the process of cure. The surrounding tissue also, after much stretching, becomes inflamed, and so brings on a fatal termination.

The direction in which cerebral abscess extends is frequently in itself also a fatal one. Following the line of least resistance, it slowly approaches the cavity of the lateral ventricles, into which there is at last a rupture. This accident may give rise to the earliest acute symptoms indicative of important disease of the brain, and then the case may be singularly perplexing.

In contrast to the insidious course of the disease when the abscess is encysted, are some instances of acute suppuration, and often sloughing where the brain becomes involved by extension of disease from the ear, or when the inflammation is secondary to chronic disease of the nose as in Cases 2 (26) and 4 (28).

There is a form of the malady where in the early stage the symptoms are not altogether unlike those of continued fever. After the febrile state has lasted for some time there may be apparent convalescence, in which either slowly or suddenly the final cerebral symptoms show themselves.

These different phases of the case probably correspond to certain stages of the disease going on in the brain;—the onset of the inflammatory action;—the gradual process of encysting;—and, lastly, the reactive inflammation from distension of the surrounding tissues. Case 11 (35) may be adduced as an illustration.

The details of the structure of the cyst are appended to Cases 12 (36) and 14 (38).

Lebert speaks of the granular character of the pus in cerebral abscess and of the small number of typical cells it contains. Similar observations were made in Case 8 (32). Such peculiarities are not noticed where the suppuration is recent, and are probably the result of secondary changes in the contents of the abscess. The same author in the article referred to, also notices the fact that abscess generally forms in the white substance, rarely in the gray, though the latter is the more vascular. This is confirmed by all experience.

The time within which a cyst may form in the brain is shown in the following case, which lately occurred under the care of my colleague, Mr. Birkett. A boy, æt. 14, whilst working in a factory received a backhanded blow from a hammer, causing compound fracture of the skull. He lay a day or two insensible, and then slowly recovered consciousness. On the sixth day from the accident, there was spasmodic twitching of the right side of the face, and loss of the use of the right arm. He was bled with relief, and two days after could grasp with the hand, but some degree of paralysis of the right side remained. At the end of three weeks the wound had progressed favorably and was nearly healed, but the boy was in a listless state. At the beginning of the fifth week he began to vomit his food, and complained of pain in the head. These symptoms were soon followed by drowsiness without any marked paralysis. He seldom spoke, but took food readily, seeming to live as a mere vegetable. He died eleven weeks from the accident. For the post-mortem details I am indebted to Dr. Wilks's report. Anterior to the point of injury, so that a trochar passed in would not have reached it, was an encysted abscess, containing about an ounce and half of pus of the usual character but not fetid. The cyst was an eighth of an inch thick, of a fibro-cellular structure, and so strong that by placing the finger in its cavity it was supported entire, whilst the brain-substance fell from it. The surrounding portions of the anterior and middle lobes were gray and ochrey, and contained abundance of granule-cells. The cyst was surrounded on all sides to the thickness of an inch by the cerebral substance. Considering the youth and previous health of the patient, it is probable we have here a fair limit, at least in one direction, of the time necessary for the formation of such a cyst. The shortest period we can infer from it is seven weeks, it was probably nearer ten. The subject has a special value in etiology, since proof of the duration of an abscess may indirectly become proof of its cause, as may be shown in the following examples: In Case 12 (36), a girl who had long been the subject of chronic suppuration in the chest, sickened with variola on the 27th January. On the 16th February she was convalescing favorably. On the 17th she became delirious and comatose, and died on the 22nd, with several encysted abscesses in the brain; the walls of the cysts being strong and vascular. It was a question whether these abscesses were the result of pyæmia, set up during the maturation of the variolous pustules, or secondary to the chest affection. The organisation of the cysts negatived the former assumption, since it was not probable such structures could develop within fourteen days. Case 4 (28) is a converse instance. The patient had a severe fall from a carriage a year and a half before his death; he had also had polypus nasi for some time, and was exposed to severe cold just previous to the onset of the cerebral symptoms. There was no trace of a cyst, but the brain-tissue which surrounded the pus was softened and vascular, as in acute inflammation. Opinions were divided as to the cause of the abscess, whether it was due to the fall or to the chronic disease of the nasal mucous membrane. The absence of any limiting wall to the abscess, and the condition of the brain-tissue around, entirely forbad the supposition that it had been so long latent as a year and a half. On the contrary, taken in connection with the recent exudation over the sella turcica, these conditions were decidedly indicative of its relation to the disease in the nose. The medico-legal bearings of the subject are obvious. Lebert gives the following details.[1] After stating that the duration of acute abscess is from three to twenty-four days, he says, "Lallemand asserts that in one instance he found a soft limiting membrane as early as the thirteenth day. In two cases it was from the twenty-second to the twenty-fourth day, so that we may place the beginning of the formation of a cyst about the third and fourth week. The greatest number are found between the thirtieth and sixtieth days, in the following proportion: In two-thirds of the collected cases which could be used in the calculation two had a duration of thirty-two days; one of thirty-five days; one of thirty-seven days; two of forty-two days; one of fifty days; one of fifty-three days; one of fifty-five days; and three of sixty days. Within this period firm, organised cysts were met with, and the later the period the more readily were they separated from the surrounding brain-substance. There were lastly two cases in the third month, one of seventy-five days' and one of ninety days' duration. And last of all a case which lasted 105 days." To this last may be added Case 13 (37), where eight months probably elapsed between the origin and final issue of the case. The conditions under which cerebral abscess occurs would lead us to anticipate great variety in its symptoms. We have seen that the disease may be from the beginning acute and rapidly progressive. In such cases the symptoms have for the most part a corresponding intensity. The most striking examples arise, as before-mentioned, from chronic disease of the ear or of the nose, in otherwise healthy subjects. Headache, often intolerably severe, is the most prominent feature. "I never wished so much to be well as I now do," said a strong man in the agony of his sufferings in one of these cases (Case 4 (28)). The headache is generally frontal, frequently more severe over the brow on the side of the abscess, but in this there is no uniformity. It is remarkable that, though there may be acute abscess, and sloughing brain and dura mater over an extensively carious temporal bone,

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[2] Op. citat., p. 100.

yet the patient may make no such complaint of local pain as to draw attention to the source of the disease. No doubt many cases from beginning to end are from this cause involved in obscurity. In 1853, Henry D—, æt. 25, was admitted under my care into the clinical ward, suffering with intense frontal headache, which he attributed to anxiety and sleepless nights. The pupils were natural. Respiration 28. Pulse 76. Tongue rather dry, with brownish fur in the centre. Bowels regular. There was no heat of the head nor any injection of the conjunctiva. He moved all his limbs freely. For three days there was no other symptom of cerebral disease, but the intense pain in the forehead. He denied that it was more on one side than the other. On the fourth day there was transient delirium, and an occasional effort to vomit. At 4 p.m., on the sixth day, the patient died suddenly and unexpectedly. On the superior part of the right petrous bone, the dura mater was destroyed by sloughing, over a large carious opening in the roof of the tympanum. In the adjacent part of the middle lobe of the brain, there was an abscess containing two ounces of fetid pus. It extended inwards to the descending corner of the lateral ventricle. The brain-tissue around was yellowish, ecchymosed, and softened. It was a matter of surprise to all who saw the case that, with such extensive local injury, beginning from the coverings of the brain, the patient should have made no complaint of pain at the part, but should have referred all his sufferings to the forehead.

In some acute cases a convulsion marks the onset of the inflammatory process.

The frequent latency of the disease and the causes which favour such a course have been alluded to. There may be absolutely no symptoms, and the abscess be only accidentally discovered after the death of the patient from some other cause. This latency, as before remarked, is for the most part only up to a certain point, and then the symptoms attending the accidents of the abscess are suddenly or slowly produced. These vary with the changes in the nervous substance. A local spasm or convulsion ; a repetition of convulsions ending in paralysis with or without insensibility ;

sudden paralysis without convulsion; drowsiness gradually increasing to stupor and coma, are some of the varieties.

The beginning of the symptoms from rupture of the abscess into the lateral ventricles has been noticed.

The attendant headache differs from the headache of tumour in being less paroxysmal, and of shorter duration before complications occur with it. The difference of this symptom in the two diseases, tumour and abscess, is to be viewed in relation to the different seats of the two lesions, and to their secondary influence on the brain-tissues. A large proportion of tumours arise in the bones and membranes. Abscess, on the contrary, principally affects the medullary substance of the brain. This is shown by a comparison of the following tables: that of the seat of tumours is from Lebert;[1] the other includes cases of abscess from Abercrombie and Lebert, and those subjoined by myself, excluding from the list the cases of abscess depending upon disease in the ear or in the nose or on other local causes which might determine its site.

Origin of Tumours affecting the Brain and its Membranes.
(Lebert.)

A. SINGLE TUMOURS—		
1. Beginning in the bones		3
2. Beginning in the membranes—		
Convex surface	13	40
Base	22	
Falx cerebri	3	
Tentorium cerebelli	2	
3. Cerebral substance—		
Convex surface of the hemispheres	17	36
Deep parts of the hemispheres	4	
Protuberance and medulla oblongata	8	
Cerebellum	4	
Pituitary gland	3	
B. MANY TUMOURS—		
Bones only	2	15
Membranes only	2	
Cerebral substance only	6	
Bones and membranes	2	
Membranes and cerebral substance	3	
		94

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[1] "Ueber Krebs und die mit Krebs verwechselten Geschwülste im Gehirn und seinen Hüllen," 'Archiv für Pathologische Anatomie,' &c., Bd. iii, 475.

Seat of Cerebral Abscess not depending upon Disease of the Ear or other parts adjacent to the Brain.

In medullary substance of the hemispheres	15
In corpus striatum	2
In optic thalamus and posterior lobe	1
In medulla oblongata with scrofulous deposit	1
In cerebellum	1
	20

It will be thus seen that in ninety-one cases of tumour (excluding the three cases of tumour of the pituitary gland), the bones or membranes were in fifty-two implicated from the outset of the disease, and that in ten at most was the tumour limited to the deeper parts of the hemispheres. Abscess, on the contrary, is very rare between the membranes, and then only when it arises from local disease of the bone, whilst in fifteen out of twenty cases it was seated in the substance of the hemispheres. It is obvious why, in such an estimate as this, the cases of abscess from diseases of the ear, which are so common, should be excluded; for, although in some cases as remarked below, the abscess of the brain which follows is less from contiguity than from secondary processes, which operate at a distance, still it is difficult in these cases to exclude localising influences. It is necessary thus to explain, or it might be a matter of surprise that the above table was so limited in its numbers.

The chronic and neuralgic character of the headache in tumour may be in part due to the inequality of its growth as well as to its seat. Abscess would produce a gradual and uniform pressure, which tumour would not.

When a patient is debilitated, the symptoms of suppuration of the brain may be as it were stifled in the general oppression of the nervous system, and the only notice of cerebral lesion, may be paralysis (often accidentally discovered), or the occurrence of a gradually deepening coma.

The cases of suppuration in the brain which in their general aspect, simulate continued fever, present, on a nearer scrutiny, many distinctive differences, such as occasional vomiting, constipation, contracted abdomen, vertigo, headache, more continued and severe than in fever; a slow, full, and sometimes intermittent pulse; impatience of disturbance, &c.

For the diagnosis of cerebral abscess it is obvious that we cannot hope to find any pathognomonic symptom. Whether the brain-tissue suffer from tumour or abscess, or be otherwise compressed and disorganised, we may in turn, and in different cases, expect to have headache, convulsion, drowsiness, paralysis, and coma. Such symptoms considered individually, or as one may say statistically, avail but little in determining the conditions which give rise to them, no more indeed than an enumeration of the letters and words of an inscription, towards its decipherment. It is their order and duration which gives them their characteristic importance.

The headache of abscess is different both in character and duration from that symptomatic of tumour. It is rarely paroxysmal and neuralgic, as it is in tumour, but more general and uniform in its expression, as well as more sudden in its rise and acute in its progress. Tumours, as we have seen, frequently affect the membranes, and often have a long chronic course; at the onset the pain is also more limited, or takes the course of particular lines of nervous distribution.

Our knowledge of the seat and character of such growths no doubt helps us to appreciate better these degrees of difference, which, like different accents on a word, give a variety of meanings to the same symptom.

General convulsion with insensibility is in itself of but little value in the diagnosis of any brain disease. In abscess it probably occurs only at two stages: at the onset of acute changes in the nervous tissue, especially if such changes begin in the vicinity of the membranes; and at a later period, if the abscess bursts inwards into the lateral ventricle, or extends outwards to the surface. Amongst the subjoined cases is one where the abscess formed between the membranes under the posterior lobe of the right hemisphere. There convulsions occurred early.

Limited convulsion without insensibility has far greater value, not as being absolutely distinctive, since the same often occurs with tumour, but as being to a great extent indicative of local disease; the character of which is to be determined by concomitant conditions and by the other symptoms. Case 13 (37) is such an instance, the clonic convulsion of the right arm without any affection of the consciousness, viewed in relation to the chronic suppuration in the chest, was an indication upon which the diagnosis was made of the presence of abscess in the brain. Abercrombie has described the remarkable course of these cases: the local clonic spasm without insensibility; the gradual supervention of epileptiform convulsion; the paralysis which follows indicating extension of irritation and pressure, more rapid in their course in abscess than in tumour.

Though convulsion may be the first symptom of both tumour and abscess, there is generally this difference in the course of the two diseases; after convulsion with abscess, the recovery is more or less imperfect, and the patient remains drowsy and oppressed; whilst with tumour, on the contrary, there may be epileptiform convulsions for a considerable time, the patient in the intervals of the seizures recovering almost, if not quite, his usual health.

The pathological order of the symptoms in abscess, as deduced from observation, is headache, local or general convulsion, drowsiness, paralysis, coma. As might have been anticipated, there is no such constancy in the presence or intensity of these phenomena as to make them unequivocal in their indication; and hence, at the bedside, it is often only by collateral circumstances, and by the known associations of the malady, that we are able to complete the diagnosis.

If it be true, as I believe it is, that with the exception of suppuration produced by scrofulous deposit, idiopathic abscess of the brain does not occur, and that, with the exception of that which follows direct injury, it is a secondary result of the suppurative process in some distant part, or of chronic disease about the head, then it is obvious that a scrutiny of every organ is the first step in the diagnosis, or that at least without which the diagnosis cannot be completed. It may often be difficult to trace the suppurative tendency to its source. In one of the cases given below nothing could be more obscure during life, and after death it was only with some labour that it was ultimately made out to be dependent upon chronic suppuration of the mesenteric glands from ileitis in a debilitated subject. In a case given by Dr. Bright, whitlow was the source of general pyæmia and abscess of the brain. In another case referred to by Lebert, the drawing of a tooth was the first step towards the morbid process. This was followed by inflammation of the upper jaw, ophthalmia, and acute cerebral abscess.[1]

The general symptoms attendant upon the formation and presence of abscess in the brain occur equally in hydrocephalus. In both diseases, acute changes may be going on within the cranium, though the head and extremities remain cool. The altered respiratory rhythm; the slow, occasionally intermittent, pulse; sluggish pupils; vomiting and constipation are evidence only of an oppressed medulla oblongata, which may arise from many causes.

Rigors are not amongst the constant symptoms. They appear to be more frequent in inflammation of the lateral sinus, or when pus collects between the dura mater and bone. The nervous tissue yields readily, and hence, probably, the tension which excites rigor is not commonly produced by the formation of pus in its substance.

Our present knowledge of therapeutics leaves us hopeless when suppuration has already taken place in the brain. The practical lesson of a large number of these cases is little more than *principiis obsta*. There is no doubt a bad and a good treatment in the most desperate cases. Could we be sure of the existence of abscess, or of the acute or chronic processes leading to it, our treatment would certainly often be very different from that commonly resorted to. Diagnosis must, however, improve before much can be attempted, and even when our insight shall have become perfect, the peculiarly unfavorable conditions of the disease will remain.

There are no sure criteria of the seat of the suppuration even whilst there is the strongest probability of its existence. The following cases, and the recorded experience of others, show that there may be pain in the forehead, with abscess in the cerebellum, pain in one side of the head, whilst the abscess is in the other hemisphere, and even no symptom but drowsiness, though suppuration is extensive. The not unfrequent occurrence of more than one abscess is also a further complication of the diagnosis. Even where abscess follows injury to the scalp, it is not so entirely under the external wound as to make us sure of evacuating the pus by an incision. Clinical experience shows, however, that the brain-tissue will bear more mechanical interference than might have been supposed, and encourages the hope that as knowledge increases even here, our power may increase with it. Detmold's case,[1] notorious from the doubts as to its veracity and the subsequent testimony[1] to its truth is a striking illustration. Though life was not ultimately saved, it was prolonged by the surgical interference, and for a time consciousness and reason were quite restored. It might be of the highest importance in a similar case if only this could be effected.

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[1] A table, originally inserted here, of the principle symptoms and causes in the sixteen cases of abscess of the brain, recorded in detail in this paper (p. 17 *seq.*), has been omitted, as it is included in the article written for Reynold's 'System of Medicine,' *see* pp. 65—84, Nos. 25—40. Ed.

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[1] The following is an abstract of this remarkable case. A healthy man, forty years of age, received, on July 14th, 1849, a severe blow from a piece of machinery, causing compound fracture of the left frontal bone. He went on well for three weeks, when symptoms indicating pressure came on. These were relieved by removing some pieces of loose bone from the wound. Nine weeks after the accident he complained of headache and became drowsy. The following day he was in a profound stupor, from which nothing could rouse him. Pulse slow; pupils fixed. An abscess was suspected. Some fragments of bone were removed without any improvement of the symptoms. It was then that Dr. Detmold determined to attempt evacuating the pus. On the 13th of September, the cicatrices which had formed over the wound and dura mater adherent to it, were dissected off, and an incision made into the brain beneath, an inch in length and an inch and half in depth. A large quantity of pus flowed out, variously estimated by those present at from two to five ounces. The patient at once recovered consciousness, and answered distinctly that he felt better. The pulse rose from 40 to 60 in the minute. The exposed part of the brain cicatrised, and the patient was able to leave his bed on the eighteenth day after the operation. Three weeks after the operation he began to lose his memory. On the 18th of October, the part from which the bone had been removed had sunk in considerably, encouraging the hope that the cavity of the abscess was closing. There was at this time a return of headache and a slight convulsion. On October 22nd, as stupor had again come on, another incision was made into the brain to the depth of an inch and a quarter without any pus being found. The next day a probe was introduced, and to the astonishment of the operator passed four and a half inches deep in the direction of the lateral ventricle, indicating that the abscess had opened into that cavity. After this the patient mended bodily, but entirely lost his memory. It was determined, as a last effort to save him, to lay open the lateral ventricle itself by an incision. This was done, and half an ounce of pus flowed

According to Lebert, there is no recorded case of cerebral abscess undergoing the process of cure. It is not improbable that, apart from the fatal tendencies of the malady, the prevalent use of mercurials, as remarked upon at the beginning of this paper, may have had its share in this unfavorable history.

Case 1 (25) is given at page 10.

Case 2 (26).—*Polypoid growth from inner wall of tympanum; destruction of membrana tympani; caries of petrous bone; sloughing of the inferior half of the middle lobe of cerebrum.*

Sarah C—, æt. 23, a healthy young woman, employed as a domestic servant, had occasional discharge from the right ear from three years and a half old, and was deaf on the same side, but as far as could be ascertained the deafness was not constant. Fourteen days before her death she began to complain of intense pain in the head, and had frequent sickness. She continued, however, to do her work until the 18th of April, 1857, when she came under the care of Mr. Stedman, through whose kindness I had the opportunity of examining the brain. The pain was then on the right side of the head and in the right ear, from which there was a purulent discharge. The pulse was slow and labouring. Her mind quite collected. The following day the symptoms were unchanged. On the 20th she had paroxysms of extreme restlessness and violent screaming, crying out "Oh, my head!" There was great cerebral oppression at times, but up to the morning of the 22nd, the day of her death, she protruded her tongue when asked to do so, and could speak distinctly. There was no convulsion nor hemiplegia. The pupils were dilated, but unequally so.

Post-mortem examination.—Thoracic and abdominal viscera perfectly healthy; no trace of tubercles. The inferior half of the middle lobe of cerebrum on right side sloughing, ash-coloured, and intolerably offensive. The sloughing extended into the descending cornu of the lateral ventricle,

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out. The patient died the same evening, seven weeks after the first opening of the abscess. On a post-mortem examination, both lateral ventricles were found to contain pus—the right a larger quantity than the left, this cavity having been in part emptied by the incision made into it before death. The incision had been carried into the roof of the anterior cornu. 'American Journal of Med. Science,' January, 1850.

[1] 'Archiv für Pathologische Anat. und Physiol.,' 1857.

and affected the body of the fornix. The thalamus and corpus striatum were superficially of an ash-grey colour, and softened. A thin offensive purulent fluid infiltrated the sloughing tissue, and was effused into the ventricles; the choroid plexus of the fourth being sodden and discoloured by it. Cerebrum healthy. The dura mater covering the roof of the tympanum, and in the situation of the lateral sinus, ash-coloured and offensive, but not perforated. Between it and the bone fibrinous exudation. The lateral sinus contained partially disorganised clots. The roof of the tympanum was destroyed by caries to the extent of a silver threepence, including a large part of the superior semicircular canal. The vestibule and semicircular canals contained pus. The cavity of the tympanum was filled with a soft cream-like pulp, which, on microscopic examination, consisted of granular matter, oil-globules, crystals of ammoniaco-magnesian phosphate, and fine acicular fatty crystals. The stapes was *in sitû*. A polypoid growth attached by a narrow pedicle to the promontory, obstructed the auditory canal. It was fibro-nuclear in structure. I am indebted to my friend, Mr. James Hinton, for the dissection of the ear.

Remarks.—If Louis's induction be of value, the absence of tubercles from the lungs was in itself evidence against the tubercular character of the disease of the ear in this case. The healthy condition of the thoracic and abdominal viscera, contrasted remarkably with the fearful ravages of the sloughing process in the brain, which had been set up by the contact of the dead and partially decomposed dura mater lying upon carious bone. The obstruction of the external meatus by the polypus appears to have been the immediate cause of the retention of the exudation in the tympanum, and thus indirectly of this destructive process. Considering the healthy character of the patient in all respects, except the disease of the ear, there is great probability that suitable local treatment would have entirely obviated the course of the malady.

Case 3 (27).—*Inflammation of mucous membrane of tympanum; caries of the chain of bones; displacement of stapes; caries of wall of meatus auditorius internus; inflammatory softening of trunks of auditory and facial nerves; abscess of cerebellum by extension of inflammation through the vein of the aquæductus vestibuli.*

Anna W—, æt. 20, was admitted into Guy's Hospital under the care of Mr. Cooper Forster, December 31st, 1856, for primary sore, ulcerating condylomata, and vaginitis. It was also observed that she had paralysis of the right facial nerve, of which she gave the following account:—"Three weeks ago one of her companions gave her a severe blow with the hand on the right side of the head. This was followed by discharge from the ear, and a week ago the face became paralysed." On repeated questioning she denied that she had anything the matter with the ear before receiving the blow. For the first few days after admission her syphilitic symptoms arrested most attention, but on the 11th January she began to complain of more severe pain in the forehead, extending to the occiput and upper part of the neck. The pain was much increased by rotating the head. She lay on the right side with her head firmly pressed between her hands. No delirium. Complained of vertigo and nausea. Frequently vomited. The day previous she had a decided rigor followed by sweating, and since that time frequent chills and heats. Face and head and extremities cold. Pulse 56, extremely small and feeble, and slightly irregular. No throbbing of carotids. Abdomen collapsed. Constipation. Fetid purulent discharge from right ear. She was treated by leeches and saline purgatives, and the two following days her symptoms remained the same. There was no delirium nor unconsciousness. Frequent moaning complaint of pain in the head, from the forehead to the occiput, and at the back of the neck. There were still frequent heats and chills, and once in the twenty-four hours a sharp rigor and sweating. The pulse remained about 60. Respiration 20, varing in depth and frequency. Early on the morning of the 14th she was taken with convulsive sobbing, without unconsciousness; this lasted for an hour or more, when she became pale, and died without a struggle.

Post-mortem examination.—Membranes and hemispheres of cerebrum healthy. In right lobe of cerebellum, near the surface, an abscess containing about six drachms of greenish mucoid pus, not fetid. The superficial wall of the abscess was adherent to the dura mater for a small space, corresponding to the entrance of the vein from the aqueductus vestibuli. The abscess was limited by a highly vascular cyst. The surrounding nervous substance was œdematous, with a few scattered exudation corpuscles amongst the loosened fibres (inflammatory œdema). The body of the fornix was diffluent and ecchymosed. The lateral ventricles contained four drachms of slightly opalescent fluid. The lateral sinus contained adherent coagula. Both portions of the seventh nerve were soft, and contained exudation corpuscles. There was also slight ecchymosis at the origin of the portio mollis in the floor of the fourth ventricle. The inflammation of the nerve-trunks had probably arisen in part by extension along the sheath, from the internal meatus, and in part from contiguity with the abscess which lay over their course. The membrana tympani in part destroyed. The handle of the malleus separated from the body of the bone by caries; the base of the stapes extensively carious and displaced from the fenestra. The semicircular canals contained a bloody fluid, but the petrous bone was not examined in a sufficiently recent state to admit of an exact account of the contents of the labyrinth. The wall of the meatus auditorius internus carious; a deposit of new bone on the upper and posterior wall of external meatus. The mastoid cells and the sulcus lateralis healthy. The lungs were free from tubercles. The thoracic and abdominal viscera healthy.

Remarks.—This patient asserted that she never had any affection of the ear before she received the blow upon it. If this was so, the crust of new bone in the meatus must have formed in the five weeks preceding her death, and whilst the ulcerative process was advancing in the tympanum. The subject has a peculiar importance in a medico-legal point of view, since it might become, as perhaps it should have been in this case, a question before a jury whether the blow was the sole cause of death, or whether the deposit of bone was evidence of antecedent chronic disease. Our knowledge of the time within which exudation ossifies does certainly not appear to contradict the statement of the patient. Admitting it to have been as she said, the immediate local treatment of such a case after the blow becomes in itself a subject of special interest. It did suggest itself at the bedside, if it were possible or not for this acute affection of the ear to have arisen from direct contact of the gonorrhœal virus.

The course of the disease in the tympanum in this case supplies another example of the uncertainty of any special diagnosis of the part of the encephalon which may suffer when the ear is affected. The caries and displacement of the stapes appear to have been followed by inflammation of a small vein of the vestibule, along which the inflammation extended to the dura mater on the posterior part of the temporal bone, and thence to the cerebellum, whilst at the same time it set up caries of the wall of the meatus internus, and passed along the trunks of the auditory and facial nerves to the floor of the fourth ventricle.

The occurrence of the facial palsy was in this case plainly owing to the extension of disease from the vestibule; it is, however, worthy of note that this form of paralysis, in affections of the ear, does not necessarily imply so formidable a complication. Not only may it occur from some temporary pressure of exudation in the bony canal, along which the nerve runs in its passage to the posterior part of the tympanum; but in scalatina, as well as in some other cases, there may be attendant glandular enlargement, compressing the nerve after it emerges from the stylo-mastoid foramen.

The chronic diseases of the ear which set up disease of the brain and its membranes, appear to be still regarded as too exclusively scrofulous in their nature. The cheesy concretion which fills the tympanum in these cases, and which is assumed to be scrofulous matter, is generally only the debris of epithelium and pus, amongst which are scattered crystals of the ammoniaco-magnesian phosphate with plates of cholesterine and fine acicular crystals, probably of a fatty character. The presence of these latter crystals, which in form are not unlike urate of soda, may have given rise to the supposition put forth by some writers on the ear, that the affections of the tympanum are often gouty, but chemical analysis gives no indication of uric acid. For the most part, these chronic diseases begin as common inflammation of the mucous membrane of the meatus, or tympanum; or when they follow upon measles or scarlatina, they soon assume the ordinary conditions of common inflammation. When the bone suffers it is by ulceration, without undergoing that change of structure which characterises scrofulous caries. A further proof of the non-scrofulous character of many of these cases is the absence of tubercles from the thoracic and abdominal viscera, even though the patients may have reached the adult period of life.

From erroneous views of the pathology of this subject, too little attention is paid to local treatment, except in the hands of the aural surgeon. Accumulated exudation is permitted to keep up irritation and favour the extension of a disease, prejudged at first sight to be scrofulous, and to need only constitutional measures for its relief, when cleanliness, occasional local depletion, and the use of astringents would remove it.

Though local treatment must be often pre-eminent, it cannot be insisted upon exclusively; for the subjects of such chronic inflammations are, for the most part, feeble and irritable, with fair hair and thin skin, and in whom nutrition often requires for its due performance the aid of medicines, as well as the most favorable hygienic conditions we can obtain.

Mr. Toynbee's remarks on this subject are so important that I cannot but quote them.[1] "There is no doubt," he says, "that, as a general rule, an attack of inflammation of the typanic mucous membrane arising in scarlatina, measles, or catarrh, subsides in persons having a healthy constitution, and it is usually in scrofulous constitutions only that the disease becomes chronic. There are, however, many exceptions to this rule, and I think I shall make it evident that it is not the scrofulous diathesis of the patient which causes the disease to advance to the brain. As a general rule, to which I have found but few exceptions, the cause of the advance of the disease inwards to the brain appears to be that matter is secreted in one or more of the cavities of the ear, from which it has only a partial egress, or in which it is entirely pent up. Sometimes it is scrofulus matter (?), at others mucus or pus; but whatever may be its mature or wherever it may be situate, I believe that its inability to escape externally is the cause of the progress inwards of the disease." Of the importance of this last remark there can be no doubt; but whether scrofulous concretion does occur under the circumstances alluded to, and if so, whether its removal would produce the same good effect as follows the removal of other accumulations, has yet, perhaps, to be proved.

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[1] Lectures, 'Medical Gazette,' 1855.

The same author has endeavoured to give greater precision to this part of pathology, by showing that each of the cavities of the ear has its particular division of the encephalon to which it communicates disease. Thus, inflammation of the external meatus will, with some special exceptions, extend to the lateral sinus and cerebellum; inflammation of the tympanum to the cerebrum, and of the labyrinth to the medulla oblongata. Although the exactness and importance of the anatomical relations pointed out cannot be overrated, and though, as Lebert, in his article on inflammation of the lateral sinus, says the observations comes from too good a source not to deserve the highest consideration, yet we must demur accepting the pathological deductions until we have more clinical proof that disease in its extension from the ear does observe such direct and exclusive routes as the anatomical relations would imply. In the above case, the disease progressed from the tympanum, in all the three directions indicated, and death occurred from abscess of the cerebellum through phlebitis of the vein of the aqueductus vestibuli. In the case of a young man, recorded by me,[1] the meatus externus and membrana tympana were healthy, yet the mastoid cells, as well as the roof of the tympanum were carious, and acute inflammation of the lateral sinus was the cause of death. In another patient, the bony meatus was carious, but disease had extended inwards and destroyed the roof of the tympanum and its posterior wall. Nor are instances wanting of acute disease of the meatus in the adult, setting up inflammation of the brain and its membranes, without implicating the mastoid cells or lateral sinus. When the course of the inflammation in any case is determined, the anatomical relations may enable us to anticipate what part of the encephalon may suffer; as, for instance, if it be towards the mastoid cells, the lateral sinus and cerebellum will become affected; if it be towards the roof of the tympanum the cerebellum will suffer; but they do not enable us to prognosticate from the beginning of a case what that course will be, whether exclusively in one direction, or generally in all. As the routes by which disease of the ear may extend to the brain are three, so are there several modes of its extension. It may be by excessive implication of all the tissues, until at length the adjacent portion of brain is involved in the lesion, or it may be by extension along the veins only to the membranes, and thence to the adjacent portion of the brain; or, lastly, abscess may occur in these cases as a secondary process, as it occurs where the primary malady is in some distant part. In this case, the abscesses may be multiple and isolated from disease in the ear by healthy tissue intervening. It is under these circumstances that the original source of the malady is apt to be overlooked and the suppuration to be regarded as idiopathic.

CASE 4 (28).—*Chronic disease of the mucous membrane of nose; acute abcesss in middle lobe of cerebrum.*

A gentleman, æt. 43, strong, of good stature, and living at the time of his illness on his own estate in the country, went to church on Sunday morning, January 20th, 1855, in apparently good health. On his way home he suddenly felt a strange sensation of lightness in the head. He felt as if lifted from the ground. As his neighbours passed him and spoke he could not comprehend what they said, and was unable to address them. He reached home safely, and soon recovered so as to be able to describe to his wife what he had felt; but shortly afterwards, when walking across the room, he staggered and fell down insensible and convulsed. The convulsion lasted a few minutes, and when he recovered he assured his friends he felt quiet well and that it was only a faintness. During the afternoon, whilst lying on the sofa, the same sensation of lightness in the head returned again, and a second time he became insensible and was convulsed. Mr. Bottomley, of Croydon, now saw him, and prescribed suitable remedies. The following day the patient appeared quite well, and would take no more medicine. On Wednesday he went to town, and on his return passed a restless night. On Thursday evening headache, of which he had had symptoms on Tuesday, returned with great violence. The pain was over the right temple, and in the occiput on the same side. I saw him first on the Friday afternoon, he was perfectly collected, complained of an agonising pain in the right temple and over the right eye. Exhibited great restlessness, now burying his head in the pillow, and now pressing it with his hands for ease. He never in his life, he said, so much wished to be well, the pain was so dreadful. Pulse 80, with an occasional intermission. No throbbing of the carotids. Skin cool. Feet cold. Temperature of head rather increased. Tongue moist and nearly clean. The bowels had been relieved during the day. Urine of a pale straw colour without deposits. Pupils small but active. No affection of sensation or motion of any part. Slight nausea. No vomiting. The history I obtained from him and his family was as follows:—He had always had excellent health until four years before. He was then living in Australia, and at the time of sheep-shearing took cold in the left eye from a *coup-d'air*. He had then severe neuralgia of the left side of the head and face, lasting three or four days, and since that time returning at intervals. For three years he had had symptoms of polypus nasi, and an attempt had been made to remove one from the right nostril. There was a free watery mucous discharge which had not diminished with the accession of the cerebral symptoms. On the day previous to his seizure he was out shooting. The weather was at the time extremely cold. After his death I learnt that at the end of the year 1853 he had a fall from his carriage, which stunned him for a short time. On the day after my visit he was seen by Dr. Jeaffreson, who found his general symptoms unchanged, except that the pain in the head had extended from the occiput to the upper part of the neck; the pulse varied in frequency, and was sometimes at 50. No vomiting. No anæsthesia, nor affection of motion of any part. Occasional and slight incoherence. He died the following morning in coma.

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[1] 'Medico-Chir. Transact.,' vol. xxxviii, p. 158.

The post-mortem examination was made by Mr. Bottomley and Mr. Edenborough, by whom I was kindly supplied with the following details, and with portions of the diseased parts of the brain.

In the posterior part of the middle lobe of the cerebrum, on the right side, was a recent abscess of the size of a small orange. It had encroached upon the deeper layer of the grey matter of the convolutions over it, but had not reached the surface. The lateral ventricles contained a small amount of puriform fluid, but no communication was traced between them and the cavity of the abscess. On removing the brain from the base of the cranium, it was found to be much softened over the sella turcica, with recent inflammatory exudation. The ethmoid bone was not observed to be carious. The interior of the nose was not examined, nor the viscera of the thorax or abdomen. The pus was without fetor, greenish, and only slightly mucoid. There was no trace of a cyst. The tissue around was softened and vascular.

Remarks.—In the absence of any direct evidence obtained by examination of the interior of the nose after death, that disease existed there which might have occasioned the abscess in the brain, we can only infer the probability of its presence from the clinical history and from the recent exudation about the sella turcica. The absence of caries of the ethmoid would not be any valid proof against it, since it is established that the blood-vessels running through a bone may be the medium of extension of disease, and especially of acute disease, before the bone-tissue becomes carious. In the diseases of the ear there are many recorded examples in proof, and Case 16 (40), given below, though not one of abscess of the brain, is an illustration of this mode of extension of disease from the nose to the contents of the cranium. When that case occurred my colleague, Dr. Wilks, spoke to me of a patient of his who had had a mucous discharge from the nares, and rather suddenly became the subject of acute meningitis. On a post-mortem examination the membranes on the under surface of the anterior lobes were adherent by recent lymph. There were no tubercles nor any other obvious exciting cause of the meningitis, and at the time the meningeal inflammation was attributed to cold, but from the history of Cases 4 (28) and 16 (40) it was probably only another instance of the course of morbid action indicated above.

CASE 5 (29).—*Chronic thickening of lining of external meatus and tympanum; ulceration of membrana tympani; cavity of tympanum filled with soft cheesy concretion; caries of petrous portion of temporal bone; inflammation of right lateral sinus; abscess on the surface of the posterior lobe of the cerebellum.*

George O—, æt. 13, one of the boys in the Licensed Victuallers' school, complained occasionally of pain in the right ear, from which there had been a purulent discharge, according to his mother's account, since he was a few months old. On the 29th May, 1854, the pain in the ear was more severe than usual, and whilst standing in his class in the afternoon he suddenly became faint. During the night he was severely convulsed, and in one of the paroxysms pus is said to have been freely discharged through the nose with immediate relief of the symptoms. June 1st, he had no evident cerebral disturbance. Pulse 96. Action of heart sharp but feeble, with irregular rhythm. Pupils dilated. Sight unaffected. On the 2nd, he made great complaint of pain across the forehead and towards the vertex, and his manner was tremulous. Slight confusion on waking. Nausea, but no vomiting. Pulse 74, irregular. Respiration 24. On the 3rd, whilst out of bed, the convulsions returned. On the 5th, after a restless and delirious night, the pulse fell to 40, with a peculiarly sharp beat. Respiration 24, very irregular in rhythm and extent, being sometimes thoracic and sometimes abdominal. Frequent alternations of flushing and pallor. Abdomen flattened. Evacuations dark and slimy. With the complaint of pain in the forehead, he also made singular complaint of formication in the toes, and up to the hip in the left side. The following day the muscles of the leg became frequently cramped, and especially those of the great toe. From this date to the 11th his symptoms underwent no important change. The pain in the head was severe, and principally referred to the forehead. On awaking from sleep his manner was frightened and confused. On the 11th he complained very much of pain and formication in the left foot, over the instep and up the leg, it was felt slightly also in the arm. The pulse ranged from 70 to 80. The respiratory rhythm was often remarkably irregular, so as to defy any trustworthy statement of it for short intervals. The skin was hot and dry. Tongue slightly furred and protruded steadily. No vomiting. The ear continued to discharge freely. Towards the morning of the 12th he became rather suddenly comatose, and died in about half an hour without convulsion.

Post-mortem examination.—The convolutions of the middle and posterior lobes of the right hemisphere of the brain were flattened, and there were traces of recent inflammatory exudation in the sulci under the arachnoid. On lifting up the posterior lobe from the tentorium an abscess was opened containing about an ounce and a half of pus. This abscess was irregularly bounded by the compressed convolutions, the falx major, the tentorium cerebelli, and the dura mater covering the petrous bone. It had not penetrated through the close arachnoid, but the subjacent brain-substance was softened, and of a faint gamboge tint. The lateral ventricles and central parts were healthy. No increase of ventricular effusion. The base of the brain and cerebellum without any traces of disease. The right lateral sinus thickened, the lower third obstructed by old coagulum, which apparently had formed slowly and was in part decolorised; the upper two thirds contained recent fibrin and pus. The external surface of the dura mater in contact with the middle and inferior third of the sulcus was much injected, and the subjacent bone roughened, only a very thin shell remaining of the posterior wall of the tympanum. The superior surface of the petrous portion forming the roof of the tympanum was ulcerated into a hole as large as a goose-quill. The tympanic cavity was full of a soft, cheesy, yellowish concretion. The lining membrane thickened, and near the opening of the Eustachian tube it was villous from delicate vascular folds. The posterior wall was excavated by ulceration. The contents of the cavity consisted of epithelium, granular matter, fat globules, and crystals of cholesterine.

The Eustachian tube was healthy. The membrana tympani in great part destroyed by ulceration. The outer bones of the chain destroyed. The lining of the meatus much thickened. The viscera of the chest and abdomen were all healthy. No traces of tubercle.

Remarks.—The thickening and villosity of the membrana tympani appeared to be due to common inflammation. In its recent state, the membrane had much the appearance presented by chronic disease of the synovial membranes, when the formation of vascular fringes attends the ulcerative process. The bone had undergone a clean process of ulcerative absorption without any preceding infiltration of its structure. The occurrence of inflammation of the lateral sinus, with ulceration of the roof of the tympanum and abscess above the tentorium, seem to show that the preponderance of the morbid action in the one direction or the other, whether towards the cerebrum or the cerebellum is, as stated above, fortuitous. The disease probably began at the superior and inner portion of the meatus, extended through the membrane to the roof and posterior part of the tympanum, and backwards to the superior part of the sulcus lateralis, the floor of which was carious for more than two thirds of an inch.

In the report of the case it is stated that a discharge of pus through the nose gave relief to the symptoms for several days. At the bedside it was thought likely that there might be some communication with the suspected suppuration within the cranium, but this was proved not to have been the case as the dura mater was not perforated. It is, therefore, probable that the accumulation in the tympanum, which may have directly compressed the dura mater through the carious opening in the roof of the cavity, was thus relieved.

CASE 6 (30).—*Chronic abscesses in liver probably set up by an attack of dysentery; recent abscess in left lung; large undefined abscess in brain.*

Thomas D—, æt. 25, sailor, was in 1853 engaged in boat service in the Burmese war, when he fell ill with fever and ague, and was invalided for seven weeks. On his passage home to England, at the end of the year 1854, he was seized with pain in the left side, occasionally extending to the right. This was attended with cough, shortness of breath, and hæmoptysis. He was admitted into Guy's Hospital, under the care of Dr. Hughes, February 14th, 1855. His symptoms at that time were cough with expectoration of thin mucus; pain on deep inspiration, and tenderness on pressure in the right hypochondrium, which was rounded and prominent; morning sweats; urine high coloured and depositing urates; no icteric tinge of skin or conjunctiva. Dulness on percussion over right mammary and lateral regions of the chest, with absence of respiratory sound, tactile vibration remaining distinct. Chronic abscess of the liver was diagnosed. In the afternoon of the 21st, whilst conversing with one of his fellow-patients, he suddenly fell down convulsed. When seen shortly afterwards the eyes were open, pupils widely dilated and fixed, breathing stertorous, with puffing of the cheeks in expiration. The tongue had been bitten. The urine passed involuntarily. The skin was sodden with perspiration. Pulse 116. These symptoms continued for several hours, and induced some who saw him to think that an extensive effusion of blood had taken place into the ventricles. In the evening five ounces of urine was drawn off by the catheter and found to be highly albuminous. The following day he was semi-comatose. Pupils dilated. Pulse 92. A pint of urine now drawn from the bladder was not coagulable by heat or nitric acid. Sp. gr. 1025, large deposits of urates on cooling. On the 23rd he had sufficiently recovered his consciousness to be aware of the pain of the blister which had been applied to the back of the neck. During the week following there was singular incoherence without delirium. His expression was vacant, and when he attempted to answer questions he frequently gave an unintelligible jargon or repeated words over and over again. There was no paralysis. On the 3rd of March he answered questions correctly but with hesitation; said his memory was returned, and that he was no longer "so silly," though at times he had vertigo and mental confusion. On the 7th he had headache and vomited; pulse 88. On the 9th the brain was again much oppressed. He could tell his name but not his age; this he tried to arrive at by enumerating the epochs of his life—as. I was so old at such a time, and so old at another. On the 12th he vomited several times. On the 14th the same mental confusion continued; instead of saying he has pain in his head, he says he has "pain in his pain." He does not recognise familiar objects though he sees them. When told to protrude his tongue he draws it back into the mouth. Hands tremulous. Pulse feeble. On the morning of the 16th he sat up in bed and drank some tea, but did not speak. An hour afterwards he was found lying on his back comatose. The right arm and leg paralysed. Both pupils dilated, the left the larger. Pulse feeble, 60. Respiration 28, entirely thoracic. Stertor with occasionally a deep sigh. During the two days following there was no important change in his symptoms. The pupils varied much within short intervals, probably from changes in the circulation, as shown by the alternate flushing and pallor of the face. Optic axes divergent; eyelids not closed. Conjunctiva on right side congested. The only traces of consciousness were in the movements of the left arm, which he often put to his head or used to replace the bed-clothes when disturbed. Died March 19th, twenty-six days from the onset of the cerebral symptoms.

Post-mortem examination.—Cranial bones, dura mater, and sinuses healthy. On removing the calvaria, an abscess was at once obvious at the surface of the posterior lobe of the left hemisphere, breaking through the cineritious substance. The cavity of the abscess was undefined. It contained about an ounce and a half of greenish mucoid pus, not fetid. The surrounding cerebral substance, including the whole of the middle lobe of the cerebrum, was softened and yellowish, from infiltration of inflammatory products. There had been hæmorrhage from the anterior and outer wall of the abscess, forming a coagulum as large as a walnut, which was partially mixed with the pus. The left lateral ventricle was full of pus, which had escaped from the abscess through a rent in the posterior cornu. The right was distended with about an ounce of clear fluid. At the base of the brain there were tender filaments of recent lymph between the two surfaces of the arachnoid. The left crus cerebri was slightly softened. Old and recent adhesions at the bases of both lungs.

An abscess of the size of an egg, filled with mucoid pus, in lower lobe of left lung; tissue around consolidated. Base of right lung in an early stage of pneumonia. Mucous membrane of colon thickened and slate coloured. In many parts well marked cicatrices of old ulcers. The calibre of the gut unequal. No existing ulceration. In right lobe of liver two chronic encysted abscesses, each as large as a duck's egg, one on the upper surface, the other in contact with the kidney. The cysts were remarkably thick and tough; they contained greenish mucoid pus, not fetid. The cyst of the deeper-seated abscess was at one part softened, and there was recent inflammation in the secreting texture adjacent.

Remarks.—In this case, one of the chronic abscesses in the liver appears to have become, by recent inflammation of its sac and of the adjacent structures, the source of secondary abscess in the lung and brain. From the nearness of the abscess to the surface, it may be inferred that the sudden and severe convulsion which was the opening symptom of the cerebral affection, was connected with membranous irritation, traces of which existed as tender filaments of adhesion between the two surfaces of the arachnoid.

The sudden seizure on the fourth day before his death may have been connected with the unusual accident of hæmorrhage into the abscess, rather than with the rupture of it into the lateral ventricle. The progress of the abscess inwards towards the posterior cornu appears to have caused ventricular effusion, which filled the right ventricle, whilst the left was full of the pus which had recently been poured into it from the abscess.

Case 7 (31).—*Encysted abscess in middle lobe of right hemisphere; undefined abscess in anterior lobe of left.*

J. S—, æt. 43, farm labourer; light hair; florid complexion; muscular. Had symptoms attributed to inflammation of the liver, from which he convalesced. On the first day of his return to work, the weather being hot, with a bright sun, September 13th, 1844, he was seized with severe pain over the left eye at the supra-orbital notch. He passed a restless night, and the following day the pain was unabated. He said he] could cover the painful part with his thumb. Pulse 72. Tongue clean. Skin cool. Bowels open. The case was treated, as one of neuralgia, with quinine. On the third day there was no improvement; he was drowsy, and the articulation defective. He was now purged, leeches were applied to the forehead, and a blister behind the ear. Calomel and antimony given every four hours. On the seventh day he was drowsy, but could be roused, and answered questions rationally. Articulation more confused—for "sleep" he said "spleep." Occasional incoherence. Sight of left eye dim. He was bled to twenty ounces. This was followed by profuse perspiration, and the pulse rose to 72. On the eighth day he was semi-comatose. Cupped to six ounces. Inunction of mercurial ointment. On the tenth day comatose. Several severe tremors (convulsions?) during the night. Pulse small and frequent. Constipation for three days. Arteriotomy to six ounces. Leeches to the head. A minim of croton oil every two hours, and ten grains of calomel with each dose. Died on the eleventh day.

Post-mortem examination.—The body that of a fine stout man. Abdominal viscera healthy, with the exception of traces of recent inflammation of liver. Left lung universally adherent, right lung only partially so. Dura mater firmly adherent to the calvaria. An abscess without definite walls, containing about three drachms of pus, in the posterior and outer parts of the left cerebral hemisphere, near the surface. The surrounding cerebral substance soft and pulpy. In the middle lobe of the right side an encysted abscess, with firm, membranous vascular walls. This had burst through the softened optic thalamus into the lateral ventricle.

Remarks.—For this case I am indebted to my friend, Mr. Brickwell, of Sawbridgeworth. The early symptoms of hepatitis may have had their origin in cerebral irritation from the presence of the encysted abscess. This must have been for some time latent. Traces of capsulation have not been found earlier than the third week. A cyst as firm and vascular as that here described would have required five or six weeks at least for its formation. It may have been latent much longer than this period. It is notoriously difficult in many cases to determine whether certain symptoms have a cerebral or a gastric origin. Biliousness is often a prominent feature in the account patients give of the early indisposition in any cerebral affection.

The intense neuralgic pain over the left eye, which ushered in the fatal attack, and which was probably a symptom of the acute abscess in the left hemisphere, was noticed also in Case 4 (28), and, at the outset, led to a similar error in diagnosis. The pain was so intense and so limited, and had followed in that case so immediately after exposure to intense cold in field-sports, that its neuralgic character was quite deceptive.

The occurrence of an acute abscess in conjunction with one encysted, is in this case ambiguous. They may have arisen from some common source not discovered at the post-mortem examination, as in the ear or nose. Or the former may have arisen as a secondary (phlebitic ?) result of the latter. This origin seems more probable, since recent changes had taken place in the cyst leading to its rupture, and to softening of the adjacent thalamus.

CASE 8 (32).—*Abscess in sheath of left rectus abdominis muscle ; abscesses in brain and cerebellum.*

Richard F—, æt. 46, labourer, applied at the hospital as an out-patient under my care 13th of November, 1854. Complained of being generally unwell and weak, and of a swelling near the superior attachment of the left rectus abdominis muscle, which on examination was found to depend upon a chronic abscess in the sheath. He had been unwell for a fortnight, but had observed the swelling only three days. He could give no account of any exciting cause. The pulse was 100. Tongue furred. Herpes about lips. Urine high coloured and albuminous. He was admitted into the hospital under the care of Dr. Addison, and the abscess was opened the same day. No symptoms occurred to arrest particular attention until the 17th, when the face was observed to be paralysed on the right side, and the same day he had a well-marked rigor. He appeared to be much debilitated, and had mild typhoid symptoms. On the 18th deglutition was difficult, and his speech indistinct. There was no decided paralysis of the extremities, but he was remarkably powerless, the left side being apparently weaker than the right. He lay from day to day in a semi-comatose condition, from which he could be partially roused. Urine and fæces passed involuntarily. Almost total inability to swallow. Skin hot. Pulse 96. Towards the end the breathing was stertorous, with loud moaning. He died November 23rd, six days after the onset of the cerebral symptoms.

Post-mortem examination.—No wound or scar on the body, except that into the sac of the abscess in the sheath of the left rectus muscle.

The integuments of the head and the calvaria healthy. The convolutions of the right hemisphere flattened. The surface of the arachnoid greasy. Exudation of plastic matter in the course of the large veins, between the convolutions. In the anterior part of the right hemisphere, near the surface, was an abscess containing about three drachms of greenish fetid mucoid pus, and on the same side, more deeply seated in the white substance, and more posteriorly, were four other smaller abscesses containing pus of the same character. They were all defined by a slight circle of injected vessels, outside of which the nervous tissue was normal. The contents, examined by the microscope, showed but few well-formed pus-cells, the larger amount consisting of granular matter. The lateral ventricles were healthy, as were also the corpora striata, thalami, crura cerebri, and pons Varolii. In the left lobe of the cerebellum was a fifth abscess, situate so near the under surface that its contents were discharged by removing this part of the brain from the cranium. The sinuses of the dura mater and the bones of the base healthy. Thoracic viscera healthy. Liver rather pale. On its superior surface, near the anterior border, the tissue was softened at two spots from recent inflammation. The splenic tissue was mottled. In neither of these viscera was there any purulent formation. Kidneys healthy. Bladder thickened. A circular stricture of the urethra about an inch from the meatus.

Remarks.—According to the prevalent theory of the secondary deposits in pyæmia, the lungs ought to have suffered before the brain in this case, provided, as was most probable, that the exciting cause of the secondary suppuration was the abscess in the sheath of the rectus abdominis. Case 9 suggests the same criticism, and, as before remarked, even more obviously than this one, since there the infected blood had to traverse both liver and lungs before it reached the brain, and produced its morbid effects. It is a characteristic example of the insidiousness of pyæmic suppuration in the brain. Slight facial paralysis was the first symptom. But for this and the occurrence of a rigor, there was little to distinguish the case from one of ventricular and sub-arachnoid effusion in a subject debilitated by chronic disease.

CASE 9 (33).—*Peyer's patches in lower portion of ileum, prominent and slate-coloured; chronic suppuration of the mesenteric glands; many abscesses in brain; abscess in right kidney and spleen.*

A gentleman, æt. 45, above the middle height, originally of rather a delicate constitution, which had been weakened by a too laborious and anxious city life, as well as by neglect of regular meals and absence from the use of all fermented liquors, was at the end of February, 1857, taken with febrile symptoms, accompanied with a rigor which returned about every twenty-four hours. The homœopathic practitioner under whose care he was supposed it to be a case of ague. In a few days the rigors became more severe, and were followed by intense neuralgic pain in the right side of the head and face. After one of these attacks, about ten days from the commencement of his symptoms, he was suddenly stricken, whilst shaking hands with a friend, with paralysis of the left side, including the face. There was no unconsciousness, and though the speech was affected he was still able to express himself so as to be understood. The rigors continued to return at irregular intervals of one, two, or three days. After each the sweating was profuse. On two occasions there was hiccough lasting for many hours. He was often drowsy, but never had headache, nor delirium, nor incoherence. At my visit, a few hours before his death, I found him perfectly collected, and on my asking him if he had pain in the head, he replied in the negative. His expression was tranquil. Pupils natural. The left arm and leg quite paralysed; the muscles flaccid and wasted. Tongue protruded to the left, its surface dry and brown. Abdomen flat. Urine and fæces had passed involuntarily for ten days. Pulse 130, feeble. On inquiry as to any cause which could have given rise to suppuration in the brain, which from the symptoms was suspected, nothing but negative evidence could be elicited. From boyhood he had often complained of a painful tightness over the forehead, and when fifteen years old had rather a severe fall. He died on the 17th of March, soon after my visit, fifteen days from the paralytic seizure and three weeks from the onset of the fever and rigors.

Post-mortem examination.—Body emaciated; muscles of left side especially wasted, and without the rigor mortis which existed on the opposite side. Under the arachnoid of the upper surface of the hemispheres several spots of ecchymosis, one as large as a florin. The middle third of the right hemisphere down to the corpus striatum was softened, leaving no distinction in the affected convolutions between the cortical and medullary structures, the whole being of a yellowish-grey colour and of the consistence of cream. Amongst the softened portion there were recent dark coagula, and a defined but not encysted abscess as large as a filbert. Throughout the medullary substance of both hemispheres there were numerous spots of suppuration from the size of a hempseed to a pea, defined but not encysted, looking like drops of pus deposited in healthy nerve-tissue. One of these abscesses, as large as a horse-bean, was seated in the inner grey nucleus of the right corpus striatum. The other parts of the brain and cerebellum were healthy. In the diplöe of the part of the calvaria removed, there were some spots of venous congestion visible through the inner table. In the centre of one larger than the rest the bone was softened, and there was pus in the cancelli. The ethmoid and temporal bones and tympana healthy. Hypostatic engorgement of lungs; heart flabby; liver healthy. Two collections of pus, each the size of a hazel nut, in the spleen, and one in the cortical portion of right kidney, The mesenteric glands corresponding to the lower portion of the ileum were enlarged and slate-coloured, and several in a state of suppuration, apparently chronic. Peyer's patches prominent, and mottled by slate-coloured dots; no ulceration. The coats of the small intestine at other parts were remarkably thin.

Remarks.—The prominence and colour of Peyer's patches in the lower part of the ileum were indications of some prior attack of ileitis. Of the occurrence of this, no other evidence could be obtained. The enlarged and suppurating glands of the mesentery corresponded to that part of the ileum so affected. The patient was a feeble person, in whom it might have been expected that local disease would extend. It is remarkable that with such extensive diffusion of the suppurative action in the brain there was never any headache nor delirium. The occurrence of the paralysis, as in many other forms of softening, was quite sudden. The rigors may have been referable to the suppuration under the capsule of the spleen and in the kidney, as well as to that in the brain.

Case 10 (34).—*Phlegmonous erysipelas of upper lip extending to the orbit; suppuration behind the globe; ulceration of the dura mater; defined abscess in anterior lobe of cerebrum.*

W. R—, æt. 16, a delicate boy employed in the stables at the South Eastern Railway, came under treatment at the beginning of May for phlegmonous erysipelas of the upper lip and right side of the face, which he ascribed to inoculation from an unsound horse. The inflammation extended to the forehead and eyelids. Abscess formed in the right orbit, and the globe protruded. There was pain in the *left side* of the head, radiating from a little behind the anterior fontanelle. A quantity of thick, creamy pus was evacuated from the orbit by puncture, and the pain over the left temple was for a time completely relieved. The eye gradually returned to its place, the chemosis abated, and almost all inflammatory appearances in it slowly subsubsided. He did not, however, recover his sight, though he gradually began to perceive a yellow-coloured light. The pain over the left side of the head returned a second time, and extended down behind the left ear and along the lower jaw. It was somewhat intermittent. No disturbance of the intellect. No paralysis, nor any symptom, except the pain, to indicate disease of the brain. He improved in flesh and strength. The headache lessened, and at times almost left him. At the end of three weeks he seemed to have regained his usual health. Whilst walking in his garden in the afternoon, about five o'clock, he complained of a dull, heavy feeling in the head, with vertigo. This went off after he had rested a little, and excited no alarm.

At 8 p.m. the same evening he suddenly fell into general convulsions, which lasted half an hour, leaving him comatose. The convulsions returned after a short interval, and he died an hour and a half after the commencement of the first fit.

Post-mortem examination.—On lifting up the brain from the base of the cranium, the membranes were found matted together on the right side over the inferior surface, near the fissure of Sylvius. The dura mater, where it covered the back of the orbit, was perforated by an opening communicating in one direction with the pus in the orbit; and in the other leading into an abscess in the middle lobe of the brain. Nearly two thirds of the middle lobe were involved in the inflammatory process. The pus was not circumscribed by a cyst, but surrounded by softened brain, outside of which there was induration, and beyond this, again, softening and hyperæmia.

Except slight flattening of the convolutions, and general fulness of the vessels (probably due to death by coma), no other morbid appearances were noted.

Remarks.—The above case was kindly sent me by my friend, Mr. Frederick Moon, under whose care it occurred. One of the points of interest in it was the seat of the pain in the head, which the patient always complained of on the left side, although the suppuration was in the right hemisphere. A somewhat similar case, as regards the origin of the suppuration, was under the care of Mr. Birkett, in May, 1854. A man, æt. 46, was admitted for slowly extending (malignant) ulceration, involving the whole side of the face and orbit. His principal symptom was headache. An encysted abscess of the size of a hen's egg, and containing fetid pus, formed in the middle lobe of the cerebrum, and extended inwards to the lateral ventricle, which contained pus. The dura mater was involved in the external ulceration, and the membranes were generally inflamed with considerable sero-purulent effusion over both hemispheres.

In this case the abscess was evidently of some date, and had formed apparently as an isolated and secondary result of the ulceration on the face, as it might have done had the primary disease been in a more distant part.

CASE 11 (35).—*Fall backwards from a cart; immediate effects overlooked; after a fortnight symptoms of continued fever; partial recovery; sudden insensibility and convulsions; hemiplegia of right side; death ten weeks after fall; abscess in left hemisphere, and in sphenoidal sinus.*

Francis L—, æt. 16, a delicate boy, was admitted into Guy's Hospital, December 23rd, 1844, with symptoms supposed to be due to an attack of typhus in a mild form. For the first fourteen days the case did not attract particular attention, and appeared to be progressing favorably. January 7th, he complained of pain in the head, which was relieved by leeching and blistering. On the 18th he was considered convalescent, and walked about the ward. He went on well until the 24th, when he was found in bed insensible. The eyes were open and fixed; mouth drawn to left side; convulsive tremor of hands and legs, followed by a succession of epileptiform convulsions, lasting for three hours, after which he lay for some time as in a profound sleep. On inquiry of his friends it was found that three weeks previous to his admission into the hospital he had slipped and fallen backwards from a cart, striking his head on the ground and being stunned for a minute or two. There was neither wound nor bruise perceptible. For a fortnight following the accident he complained of nearly constant pain in the back of the head, and was generally indisposed, but not so unwell as to prevent his following his employment. At the end of the fortnight the pain in the head was more severe, and he had chilliness, languor, and loss of appetite, and was sent to the hospital as a case of fever. On the day following the convulsions (January 25th) he was partially sensible, seemed to comprehend what was said to him, but made no attempt to speak. He had a puzzled, confused expression, but protruded the tongue when told to do so. Pupil contracted, but obedient to light. Urine and fæces passed involuntarily. During the next two days he became more sensible, and on the 28th spoke, and complained in a general way of pain in the head. On the 30th he vomited, and from 9 a.m. to 2 p.m. had a series of epileptiform convulsions, after which he lay in a state of coma. On the 31st he sat up in bed and put out the tongue when told, but did not appear to recognise his friends, nor could he be made to speak. The right arm and leg were completely paralysed. The tongue was pointed to the right; face occasionally drawn to the left. When the left arm was lifted up it remained for some time unsupported in that position, as in catalepsy. Breathing natural. Pulse feeble and quick. Gums affected by mercury. Towards morning, February 1st, he was for a short time violent and excited in manner. During the day it was noticed that the face was less paralysed, and that he could to some extent move his right arm. He answered questions in monosyllables, and talked incoherently to himself. When the tongue was protruded he seemed to forget to draw it into the mouth again. His expression was remarkably vacant. He sat up in bed by himself, and was incessantly catching at imaginary objects about his dress. A careful scrutiny was made of the head with the view of trephining, but no trace of any wound or bruise could be detected. All medicines were omitted, and the *ferrum candens* applied to the vertex. For the next few days there was no important change. He occasionally vomited, without any apparent cause, and started up in bed agitated. The pulse was about 60. He had a stupid, oppressed look, but was not insensible, and could articulate slowly, with tolerable distinctness. From the 8th he became semi-comatose, and could be only partially roused. No stertor. Pulse 60. The food placed in his mouth remained unmasticated. A succession of convulsions came on during the night of the 10th, and he died early the next morning, about ten weeks from the accident.

Post-mortem examination.—No marks of injury were perceptible either upon or within the cranium. The cerebral convolutions were flattened. The arachnoid membrane dry. The vessels congested. The lateral ventricles contained an excess of fluid. In the left hemisphere, a little below the level of the corpus callosum, was an encysted abscess, containing between two and three ounces of serous fluid, in which there was a sediment of greenish-yellow, thick pus, whilst flocculi of lymph floated above it. The wall of the abscess was formed by a well-organised false membrane, having internally a smooth mucous surface. The abscess encroached on the corpus striatum and thalamus opticus. The cerebellum and other parts of the brain healthy. The processus olivaris of the sphenoid bone was carious, and around it there was a deposit of new bone. Under this part of the cranium, in the sphenoidal sinuses, was a symmetrical abscess, encysted like that in the brain. It had no communication with the cavity of the cranium. Heart healthy. Pneumonic consolidation of the posterior parts of both lungs. Calcareous deposit in the bronchial glands. Intestines glued together by old adhesions. Other viscera healthy.

Remarks.—Injuries to the head, as in this case, are a fertile source of, so-called, idiopathic cerebral abscess. The subject presents difficulties which cannot be altogether surmounted. In the routine of life there will always be, on the one hand, a forgetfulness of such events as these, if they do not lead to immediate results, and, on the other, an acute remembrance of trifles, however absurd, which happen to be associated with the beginning of any symptom. These failings of human nature will perplex us at the bedside in proportion to our want of clinical experience in the particular cases under consideration. This alone can tell us the value of negative evidence. The peculiar course of cases like the above has been already remarked upon, and the chief points of difference from continued fever enumerated.

CASE 12 (36).—*Cirrhosis of left lung from pleuro-pneumonia in childhood; dilated bronchial tubes; encysted abscesses of brain.*

Jane T—, æt. 17, a thin, delicate girl, dark hair, rather sallow complexion, never had menstruated, was, on several occasions from the age of fourteen, an out-patient of Guy's Hospital, under my care, for cough and purulent expectoration, with occasional hæmoptysis. When three years old had measles, with pleuro-pneumonia of the left lung; this was followed by hooping-cough. Flattening and general contraction of the left chest resulted, with the ordinary signs of obliterated pulmonary tissue and dilated bronchial tubes. On the 27th of January, 1855, she sickened with variola, which assumed a semi-confluent form. In the stage of maturation the habitual cough became more severe, and the expectoration was increased. On the 14th of February she appeared to be convalescing favorably, except that her manner was rather dull and she complained of headache. During the 15th and 16th there was no noticeable change. On the 17th she became delirious and left her bed, thinking her room was on fire; her mother found her sitting on the stairs, cold and faint. After being put to bed she had a well-marked rigor, and complained of increased headache. On the 18th the left arm was observed to be paralysed; she could move the leg on the same side readily, and the face was unaffected. There had been no convulsion. Her manner was dull, but when urged to speak she could answer intelligibly. Pulse 84. Constipation. During the 20th and 21st she complained of great pain in the left leg, and early on the 22nd began to move it incessantly up and down in bed, the left arm remaining motionless. The constipation persisted, notwithstanding the repeated administration of purgatives, during three days. About noon she rather suddenly became comatose, and died the same day.

Post-mortem examination.—On removing the calvaria and dura mater, the convolutions on the right side were seen to be flattened as by pressure from within. The minute vessels of the pia mater on both sides were congested. The right lateral ventricle contained three drachms of greenish mucoid pus, without smell, which had escaped from an encysted abscess occupying the whole of the right optic thalamus, and pressing upon the adjacent corpus striatum. The body of the fornix and septum lucidum were softened and broken down. In the posterior third of the left hemisphere were two smaller encysted abscesses, containing each about a drachm and a half of pus. The wall of the cyst of the larger abscess was from a line to a line and a half thick. It consisted of fibro-cellular tissue. The inner surface smooth, like mucous membrane, highly vascular, of small, irregularly dilated, meandering veins. The brain-substance around was softened, apparently from œdema only, no exudation-cells being discovered amongst the loosened nerve-tubules. The bones of the head and the sinuses of the dura mater were healthy. The left lung was universally adherent, and only with much difficulty removed from the chest; it weighed fourteen and a half ounces, and sank in water. On section there were irregular cavities throughout it, communicating with the bronchi (dilated tubes); these were lined by a delicate membrane of a dark venous colour, continuous with the vascular lining of the bronchi. Three of them were of considerable size, one near the apex being as large as a hen's egg. The pulmonary tissue was entirely destroyed, and replaced by a dense fibrous structure, incorporated with the thickened pleura. The right lung was healthy, as were also the abdominal viscera. No trace of tubercle.

Remarks.—The latency of the abscesses in this case, and their relation to the chronic disease in the chest, have been referred to above. The occurrence of smallpox may have been a determining cause of augmented activity in the cysts, and may thus have promoted the fatal termination; but there is no probability, as before stated, that the smallpox had part in exciting the suppuration, since this must have been long antecedent to the operation of the variolous poison. This and the two following cases are instances of a similar association of chronic disease of the chest with cerebral abscess.

The doctrine of final causes, so freely quoted by modern pathologists in explanation of morbid phenomena, receives a shock from the facts of cerebral abscess. Whatever "efforts of nature" the course of abscess elsewhere may seem to show, we can recognise in its course in the brain but mechanical principles, according to which the yielding is in the direction of least resistance, and the pus thereby slowly makes its way towards the cavity of the lateral ventricles irrespective of the well-being of the individual.

The sudden accession of delirium was probably brought on in this case by such an accident. In a similar case, recorded by Abercrombie, a man, æt. 43, had for ten days complained of headache, but was still able to follow his employment. Early one morning he was seized with palsy of the left side of the face, and became unmanageable. The pupils were contracted and the eyes in perpetual motion. He made the most powerful resistance against being bled. He died on the fourth day. Three encysted abscesses were found in the brain, the largest of which had burst into the lateral ventricle.

Case 13 (37).—*Pleuro-pneumonia of the base of right lung, followed by fetid expectoration; death after three years from encysted abscess in the brain; cavity in lower lobe of right lung, with several bronchial tubes opening into it.*

A gentleman, æt. 34, tall and well proportioned, with light hair and fair complexion, was in good health until December, 1853, when, from exposure to cold, he had an attack of acute pleuro-pneumonia of the lower lobe of the right lung. In March following the symptoms returned, and he began to expectorate muco-purulent fluid of a peculiar earthy fetid odour. In a few weeks he was able to go to business, but was never again robust. The expectoration continued purulent and fetid. Exacerbations of the local chest-symptoms occurred from time to time, and were generally attended with slight hæmoptysis.

In this way he went on until March, 1856, when he had a sudden seizure, lasting for nearly two hours. The symptoms were vertigo, faintness, and loss of power on the right side; he was not unconscious. The day following he was at his business as usual, but ever after he would at times complain of *a tired feeling in the head*, and was easily fatigued. Singing at church and other loud noises distressed him. With these exceptions, however, he appeared in his usual health, and continued to perform active duties; and though I often saw him for the chest affection, he never complained of anything in the head. November 15th, about two o'clock, he was surprised by a sudden and violent chronic convulsion of the right arm, lasting for several minutes. It was so severe as to oblige him to support himself by holding the table with the other hand. He felt quite well at the time, and when the muscular action subsided went on with his duties as before. He left the warehouse at four o'clock, and was walking to the omnibus, when the right arm was similarly affected a second time for a minute or two. He still felt quite well, and took his place in the omnibus, but had not proceeded far before the movements returned a third time, now affecting slightly the muscles of the face and of the leg on the same side. After lasting as before, but a few minutes, they again left him. When I saw him at six o'clock he appeared quite well. The voluntary movements were everywhere perfect; vision and pupils natural; no headache nor trace of mental disturbance, and, with the exception of slight and transient vertigo at the time of the third return of the movements, there had been no symptom referable to the brain. He assured me he felt quite himself, and on my requesting him to walk round the room and examine the pictures and engravings on the walls, he asserted that he saw them quite naturally. He continued well until noon of the following day (November 16th), when the convulsions returned a fourth time, beginning in the same way with clonic spasms of the arm and face, on the right side, without loss of consciousness, but quickly assuming the character of a severe epileptic seizure, with insensibility. After two hours he recovered, and the next day (the 17th) wrote to his sister, and complained only of weakness. On the 18th he had another convulsion, with insensibility, followed by partial paralysis of the right arm and leg. On the 20th he slept nearly the whole day; when awake he was quite himself, except the inability to move the right arm and leg freely. Up to this time he had no headache, and no delirium. On the 21st and 22nd he continued in the same state; pulse 80; skin cool; no heat of head; perfect clearness of intellect when fully awake; slight headache towards evening; constipation. On the 24th the arm and leg were more numb and powerless; about noon the whole side was convulsed, and he became insensible. The convulsions continued, with only short intervals of quiet, until 4 p.m. After consciousness returned he remained speechless for many hours. On the 25th and 26th he was much troubled by the almost constant recurrence of the clonic convulsion of the right side, including the face, but without insensibility. After the application of leeches, a blister, and free evacuation from the bowels, he was much relieved, and slept tranquilly. On the 27th he complained of a "dead pain" in the head; there was still no febrile heat; he raised the right arm with more power; pulse 80. On the 29th the arm and leg were quite paralysed; the muscles flaccid; sensation not much diminished. By this time he had greatly emaciated; when asked if he had pain in the head, he replied slowly —"I ought hardly to say pain," and afterwards added, with a smile, that he was comfortable. The administration of an enema brought on convulsion without insensibility. On December 3rd there was great cerebral depression; he used words incoherently, asking for one thing when he meant another. There had been no convulsive movements for two days, but now and then he seemed to faint for a time; the left hand was constantly pressed to the left side of the head; the right arm and leg were perfectly paralysed, and the muscles of the face on the same side partially so; pulse 80; tongue protruded straight. On the 4th he uttered a few sentences very slowly and interruptedly, but lay for the most part in a half comatose state; took food readily. During the 4th, 5th, and 6th, he lay perfectly quiet, occasionally breathing with a little stertor, but generally so calmly that it was difficult to say whether he breathed or not; could be roused, and understood what was said to him; expressed by signs his desire to have food and the like. On the 7th the urine passed from him involuntarily, though he retained his consciousness, and once smiled faintly when his sister was near him. From his movements it was evident he suffered a good deal of pain in the head. He could not understand questions, but recognised signs; pulse 56; respiration 20; pupils equal, rather less contracted than in sleep; abdomen collapsed; several rigors, followed by spasmodic extension of the whole body. On the 8th was unable to swallow; towards evening very severe convulsions came on, and lasted several hours, when he died exhausted.

Post-mortem examination.—The integuments of the head and calvaria healthy. In the posterior lobe of the left hemisphere of the brain, on a level with the corpus callosum, there was an encysted abscess, containing two ounces of mucoid, greenish, fetid pus. The cyst was bounded externally by a thin layer of greenish brain-substance, the membranes over it were not inflamed. The wall of the cyst was about one tenth of an inch thick; it was vascular. The brain-tissue around was softened from inflammatory œdema. Slight increase of fluid in the lateral ventricles. No disease of the temporal or of the other bones at the base of the cranium. Firm pleuritic adhesions over the lower lobe of the right lung, and an irregular cavity, as large as a pullet's egg, in the pulmonary tissue. The lining membrane of this cavity was smooth and transparent, and on the proximal side perforated by several bronchial tubes. The tissue around was indurated. No trace of tubercle in any of the tissues. Abdominal viscera healthy.

Remarks.—The sudden seizure in March, though so transient that it caused no alarm, was probably indicative of the commencement of the suppurative process in the brain, which afterward pursued, during eight months, an entirely latent course.

The order of the symptoms in the final attack, was characteristic of the onset and extension of the inflammation around the old cyst of the abscess. Clonic spasm of the right arm as an isolated symptom, without any other disturbance of the nervous system, was the opening phenomenon. The day following the convulsions became epileptic. On the fourth day, after a repetition of such convulsions, the right side was left partially paralysed. The hemiplegia then gradually became complete, and the patient became drowsy and indifferent, and at length comatose. These symptoms, associated with the chronic disease in the lower lobe of the right lung, were the basis of a correct diagnosis.

CASE 14 (38).—*Encysted abscess in posterior lobe of left cerebral hemisphere; heart drawn over to right side of chest; pleuritic adhesions; external fistulous opening, and dilated bronchial tubes on the same side.*

J. H—, æt. 23, a policeman, who had been in Guy's Hospital a year before for pleurisy, was again admitted March 23rd, 1853. His symptoms were considered obscure. It was suspected that he might be labouring under chronic hydrocephalus. There was mental confusion and loss of memory. No paralysis. He was sensible until his death, which took place unexpectedly April 28th. The account he gave of his illness was, that six weeks before admission, whilst on duty, he had a sudden seizure with partial loss of consciousness, and the same day a second seizure. After a month he had a third seizure. From the time of the first attack he had headache and vomiting.

Post-mortem examination.—No disease of the cranial bones. Membranes of the brain healthy. A large abscess in the substance of the white matter of the posterior lobe of the left hemisphere, not implicating the thalamus opticus. The walls of the abscess were remarkably thick, and in many parts mottled with tortuous capillary veins. The whole cyst was easily enucleated from the surrounding cerebral substance, which was softened and of an opaque white. The walls of the cyst could be divided into three layers; the external layer was finely fibrous and rather loose, and composed of a cellular web, the remains of softened nervous tissue; the middle layer was dense and translucent; it was formed of fine, rather flattened fusiform fibres, with elongated nuclei; the inner layer, or so-called pyogenetic membrane, was opaque and brittle; it consisted of an adherent layer of nuclei and exudation cells, many of which were undergoing granular degeneration. The pus amounted to about two ounces. It was clotted and mucoid, and decidedly alkaline. It contained opaque granules and fibrinous shreds undergoing earthy infiltration. The abscess had burst into the descending cornu of the lateral ventricle. The surface of the corpus striatum and thalamus on the side of the rupture was of a dull colour, but without inflammatory exudation. Right lung universally adherent. The lower portion of the pleura thickened, and in it a fistulous canal opening externally, but not communicating internally with the lung. Pulmonary tissue compressed, the tubes dilated. Heart drawn over to the right side by adhesions of the pericardium to the lung. Left lung partially adherent.

Remarks.—There cannot in the nature of the case be any symptoms pathognomonic of abscess of the brain. It is often only from the collateral circumstances that we can rightly estimate the nature of the cerebral disorder. In this case the sudden seizure with partial loss of consciousness, if not epileptic, should have caused a suspicion of some local disease in the brain. The same symptom has often been noticed in cases of tumour as well as of abscess. The differential diagnosis would rest upon the absence of pain preceding and following the seizure; the rapid progress of the symptoms, marked by weeks, rather than by months; the character of the cerebral oppression; and the local conditions of the chest. The frequent absence of rigors in cerebral suppuration has been already noticed. Although the contents of the abscess were undergoing the earthy change there was a gradual extension towards the lateral ventricles.

CASE 15 (39).—*Inflammatory cysts, with surrounding solid exudation in anterior part of right hemisphere of brain, probably excited by* contre-coup.

William S—, æt. 40, a tall, athletic man, of rather free habits, though not a drunkard, by occupation a mill-wright, began to suffer with severe headache in the spring of 1854. The pain was principally across the forehead, and at times so severe, that, to use his own expression, he thought he should go out of his mind. He continued at his work until the end of November, when his memory became impaired; he had transient attacks of unconsciousness, and at other times lost his sight for half an hour or so. He had frequently double vision, and was troubled by dark, irregular forms creeping before him. From this time he had vomiting and constipation, and began to emaciate. January 5th, 1855, he was admitted into Guy's Hospital under the care of Dr. Barlow. His manner was dull, he complained of constant pain in the head, aggravated in paroxysms. There was no paralysis, but his movements were sluggish, and he could not stand long without feeling faint. Vision impaired. Pupils dilated. Urine scanty, depositing phosphates largely when heated. His wife gave an account of a severe fall which he had about Christmas, 1853, by slipping upon the pavement and striking the back of his head. He was stunned at the time, but the effect soon passed off, and the accident was forgotten. He remained in the hospital until the 24th of February. On several occasions he had seizures in which he lost all muscular power; these he called faintings. At the beginning of April he was readmitted into the hospital. He was then totally blind. He could neither walk nor stand, but could move his limbs, though he did so tardily, especially on the left side, and remarked that "they did not feel the right thing." The left seventh nerve was partially paralysed. Tongue projected straight, covered with a thick creamy fur. Left to himself he lay continually as if asleep. When aroused he answered questions slowly, in a loud, monotonous voice, interrupted by an occasional yawn, or dozed off in the middle of a sentence. "Has an awful headache, and that's all his complaint." Pupils widely dilated and unaffected by light. Urine and fæces the last few weeks passed involuntarily. Occasional returns of the fainting seizures. He took readily whatever food was put into his mouth, but was too dull to feed himself. Deglutition perfect. Pulse 64. Respiration 16, tranquil. He lay in a semi-comatose state until his death on the 26th. The right pupil was large and fixed, the left smaller and unaffected by light, but spontaneously dilated and contracted. There were no convulsions. A few days previous to death several bullæ formed on the legs and ankles, and he became slightly jaundiced.

Post-mortem examination.—The wasted body of a strong man. Integuments of a faint yellow colour. A few large blebs on legs and feet. Head very large and round, measuring thirty-three inches in circumference. Calvaria very thick, dense, and heavy. The whole inner surface was vascular, and roughened by ossific granulations. The outer surface of the dura mater was roughened in correspondence with the bone. Arachnoid slightly opaque. The anterior lobe of right hemisphere swollen and pressing over on the left. The convolutions flattened. Immediately below the surface a cyst the size of an egg, and to its inner side a smaller one. They contained a clear, yellow, mucoid fluid, with opaque particles floating in it. These consisted of fatty granules, loosely collected together or contained in large, regular cells. The larger cyst was round and circumscribed, except on the inner side, where it was connected with the smaller one; this was not so well defined. The lining of the larger cyst was smooth, with numerous small veins ramifying in it. The walls consisted of yellow solid exudation, part fibrillated and part granular, containing exudation-cells and oil-globules scattered or grouped tgether. The walls of the smaller cyst were flocculent, but presented the same microscopic indications of inflammatory deposit undergoing fatty degeneration. The brain-tissue around was yellow and soft with vascular striæ. The cysts pressed on the right crus cerebri, on the second, third, and fourth nerves of the same side, and on the opposite hemisphere. The inflammatory softening extended to the middle lobe and to the outer side of the corpus striatum. The thoracic and abdominal viscera healthy, with the exception of recent hæmorrhagic infarction of the spleen and of the mucous membrane of lesser curvature of stomach.

Remarks.—This case is given as a connecting link between tumour and abscess. The symptoms and chronic course of the case were such as occur in tumour; obstinate headache continuing for months, followed by double vision and partial amaurosis, then vomiting, constipation, and cerebral oppression. The mechanical action of the cysts on the surrounding structures would obviously be nearly as in tumour. The difference of the structure of such cysts and of their contents from those of abscess, is probably one only of degree, depending upon the activity of the morbid changes. The operation of *contre-coup* was favoured by the height of the man's stature, and the size of his cranium ; circumstances, apparently trifling in themselves, but not to be overlooked in estimating the effects of such a fall as he he had.

CASE 16 (40).—*Chronic disease of mucous membrane of nose; partial absorption of horizontal plates of ethmoid; inflammation of under surface of anterior lobe of brain on right side.*

Thomas W—, æt. 42, employed at the tap of a public house, and of very intemperate habits. For an uncertain period he had been troubled with mucous discharges from the nose, and with vertigo and headache for five weeks. Ten days before admission into Guy's Hospital, March 29th, 1855, he went to bed in his usual health, and the next morning was found in bed insensible. He remained in this state, recovering apparently only the slightest glimmering of consciousness, and at the best not able to recognise his nearest friends. When first visited, he lay supine and apparently insensible. The eyes were open, and directed slightly to the right side. When addressed in the loudest voice, no impression was made upon him. Both pupils were dilated, the right the larger, and slightly active. Left arm and leg motionless. With the right hand he so incessantly rubbed the chest and groins that it was necessary to muffle it to prevent further abrasion of the skin, which he had thus already produced. Respiration 16, stertorous, and with puffing out of the cheeks. Pulse 112. There was no alteration of symptoms. Towards the end the breathing became quick, short, and entirely thoracic. He died April 1st.

Post-mortem examination.—The head only was examined. With the exception of a few slight opacities on the arachnoid, the membranes on the upper surface of the brain were healthy. The arteries and veins were full of dark blood. The lateral ventricles were dilated, and contained about an ounce of clear fluid. Their surfare (ependyma) granular. Fornix and septum lucidum firm. On the under surface of the anterior lobe of the cerebrum, on the right side, from the olfactory bulb backwards to the fissure of Sylvius, the membranes were adherent and thickened, and the convolutions softened. The olfactory bulb itself was slightly enlarged, and converted into a firm yellowish mass. The grey matter of the convolutions was eroded; the white substance of a yellowish-grey colour. The softening skirted the edge of the longitudinal fissure backwards to the fissure of Sylvius, so as to affect the under surface of the corpus striatum. The horizontal plate of the ethmoid was very thin, but not carious. The mucous membrane lining the nasal cavities hyperæmic, and on the right superior turbinated bone it presented an uneven granular surface, but no ulceration.

Remarks.—This case is introduced as an appendix to Case 4 (28), to illustrate the extension of disease from the mucous membrane of the nose to the membranes and substance of the brain without caries of the bone. There was no ulceration of the mucous membrane. We may thence conclude that the changes in the capillaries through which the morbid action is distributed, may be independent of any destructive action in the tissues. In gonorrhœa, the capillaries and their contents propagate the morbid action without ulceration. General phlebitis may occur from simple sprain of a joint without any lesion of the skin or tearing of deeper parts.

ABSCESS OF THE BRAIN.[1]

BY

WILLIAM W. GULL, M.D., AND HENRY G. SUTTON, M.B.

Abscess of the Brain is comparatively a rare disease, and it falls to the lot of no man to see a great many cases. We have collected seventy-six cases in all from various sources, and the details in this paper are based upon these records. Many of the cases have not before been published. We have arranged the different parts of this subject in the following order:—A description of the various conditions that are known to give rise to cerebral abscess, the morbid anatomy, the symptoms, pathology, diagnosis, and treatment.

Suppurative inflammation of the brain may be caused by injury to the head, especially where the skull is fractured and the brain contused. Mr. Prescott Hewitt says:—"All traumatic inflammation of the brain substance may end in suppuration and abscess."

Cerebral abscess may follow a penetrating wound of the brain substance, by a knife, by a splinter of wood, or by some sharp instrument being forced through the skull (Case 74).

Abscess of the brain may follow a fracture of the skull where there is no displacement of the bone; acute suppurative inflammation of the membranes and brain substance being set up by the injury (Case 1). In many cases, caused by fracture of the skull, the abscess in the brain is seated immediately under the injured bone, and close to the surface of the hemisphere. In others the abscess is not seated near the surface; for instance, a person may receive a fracture of the skull, symptoms of compression may set in, and the skull may be, in consequence, trephined; the portions of depressed bone may be removed, and the patient go out of the hospital apparently well. But after a few weeks or months, cerebral symptoms may again appear, and the patient may die, and the autopsy reveal an encysted abscess embedded in the substance of the brain, and seated at some distance from the surface (Case 2).

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[1] Reprinted from Reynold's 'System of Medicine,' vol. ii, 1868, by permission of Dr. Russell Reynolds and Messrs. Macmillan and Co.

Cerebral abscess may follow an injury to the skull, where there is no fracture of the latter, and with (Cases 15 and 53), or even without, a scalp wound. In such cases the injury excites inflammation and suppuration of the diplöe of the bone, and the suppuration extends and involves the brain.

Cerebral abscess may follow contusion, or, as it is sometimes expressed, concussion of the brain, without there being any fracture or other discoverable injury to the skull. Mr. Prescott Hewitt says that he has seen two cases of this kind, and the abscesses were large.[1]

This is a very important class of cases, for it probably embraces not a few of the so-called idiopathic abscesses of the brain.

In two of our cases, abscess was found in the brain, though in neither was there any evidence to show that the skull had been fractured or otherwise injured. With both patients the symptoms followed directly after the injury; one had a fit on the same day as the accident, and the other suffered from almost constant pain in the head for a fortnight after the accident, and was otherwise generally indisposed. The abscesses were encysted in both instances, and, during the time they were forming, there were symptoms indicative of cerebral disease, although, in the second case, the symptoms were, for a while, obscure. One patient died seven weeks, and the other three months, after the accident.

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[1] 'Holmes' Surgery,' vol. ii, p. 185.

Cases might be given to show that abscess may follow injury to the head, without any fracture or other discoverable injury to the skull (Cases 7, 13, 20, 35, 39, 43); and the abscess may remain latent for months or even longer.

One of the commonest causes of cerebral abscess is disease of the internal ear. The clinical history of this class of cases is usually as follows:—The patient has a discharge from the ear for some time—for months—and, in many cases, for years; the discharge being continuous or intermittent. It is common to hear it said that the discharge began in childhood, after an attack of measles, scarlatina, or smallpox; and since has returned, more or less. With the discharge there is often deafness and pain in the ear, but more often the patient makes no complaint of either. In some cases the discharge is very offensive, and has been so for some time past. The extension of the disease to the brain is often very insidious. There may be no indications that the brain has become seriously involved until acute symptoms set in a few days before death. Very often the first sign is a great increase of the pain in the ear. The pain is often very severe, and comes on in paroxysms, so violent in some cases, that the sufferer screams with it. Occasionally the acute mischief in the brain is ushered in with rigors; at other times with nausea and vomiting.

Sometimes an epileptiform convulsion ushers in the acute symptoms, and a few days after this the convulsion is repeated, and followed by hemiplegia.

The accession of acute symptoms appears, in many cases, to correspond with the commencement of acute inflammatory softening, either primarily in healthy brain, or secondarily around an old abscess. Then the skin becomes hot, the pulse quick, tongue dry and parched, great prostration, drowsiness and stupor set in. Such symptoms as resemble continued fever, and have been mistaken for it in some cases. The discharge from the ear varies very much during the acute symptoms. It is common for it to subside, or even entirely to disappear.

Chronic changes, dependent upon disease of the internal ear, may be insidiously going on in the brain substance, without there being any symptoms of cerebral disease.

Mr. Toynbee was of opinion that the inflammation extends to the brain, from the pus not escaping from the cavity of the tympanum externally. He says: "So long as there is a free exit for the discharge, I believe the disease rarely extends to the brain."[1] He also remarks: "In all fatal cases the discharge has been deprived of a free egress." Mr. Toynbee further states, in cases where the disease attacks the mastoid cells in early life, the cerebrum is the part of the brain which is most likely to suffer, while in later periods of life the cerebellum is the part most generally affected. Long experience has clearly shown that, when disease of the internal ear has gone on for a long time, the temporal bone is very liable to become diseased. When the patient dies with cerebral symptoms, it is common to find caries of the petrous, or mastoid portion of the temporal bone. It is also common to find suppurative inflammation of the dura mater covering the diseased bone, with or without sloughing of that membrane. There is, in some cases, no direct extension of the disease from the bone to the contiguous parts. In such cases the bone, membranes, and surface of the brain, are healthy. A portion of healthy brain may lie between the abscess and the bone. The diseased action is considered to extend by a vein. It is rare to find abscess of the brain following acute disease of the ear; but one case is alluded to by Mr. Toynbee.

In cases of chronic disease of the ear, the causes of the acute brain mischief are various. A blow on the head, violent exercise, or other depressing influence; also cold air, or some irritating application, is sufficient to engraft acute changes upon the chronic disease.

Cerebral abscess may be associated with, and apparently dependent upon, chronic disease in the lungs; but in two of our cases the morbid appearances were such as to indicate acute changes in the lungs, extending, however, over several weeks (Cases 9 and 57).

In a case that occurred in St. Bartholomew's Hospital, the lung presented the appearance of acute pneumonia in the third stage; but the symptoms indicated that the disease had been going on about two months and ten days (Case 9).

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[1] *Vide* 'Diseases of the Ear,' by Mr. Toynbee, p. 303.

In all the other cases, which have come under our notice, the morbid changes in the chest had evidently been going on several months and even years (Case 38). In one there was a large suppurating chronic empyema (Cases 10 and 11). In another there was a large cavity at the apex of the right lung, which was firmly adherent to the chest walls by a thick layer of indurated tissue. Another patient had had flattening and general contraction of the left chest for years, signs of dilated bronchial tubes, and of disease in the left lung (Cases 36 and 38).

Suppuration in any part of the body may give rise to secondary abscess in the brain. In one of our cases there was an abscess in the sheath of the left rectus abdominis muscle, and several abscesses without cyst in the brain (Case 32). In this case it is instructive to notice that the lungs, the common seat of pyæmic abscesses, did not contain any abscesses, nor were there any in the liver or spleen. In another case there were pyæmic abscesses in the brain (Case 33), apparently the result of chronic suppuration of a mesenteric gland, and co-existing recent abscesses in the spleen and kidney. In a case of acute necrosis of the tibia (Case 4), which occurred in St. Thomas's Hospital, there were numerous abscesses in the brain, and pyæmic abscess in the lungs, liver, and spleen. In a case given by Dr. Bright, a whitlow was the source of general pyæmia and abscess of the brain. In another case, referred to by Lebert, the drawing of a tooth was follow by inflammation of the upper part of the face and cerebral abscess. Dysentery was the cause in one instance (Case 25); abscess near the uterus (Case 8); suppuration in the Fallopian tube (Case 75); carcinoma of the face (Case 50); abscess in the liver (Case 51); and the phagedænic ulceration, following amputation of the breast (Case 56), were the causes in other cases. Dr. Ogle relates a case of secondary purulent deposit in the brain, apparently the result of ulceration of the cœcal appendage. There is also another recorded case following amputation of the forearm.[1]

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[1] From analogy we should expect that an hydatid tumour, or so-called strumous deposit in the brain, would cause abscess. We have, however, no

In chronic disease of the bones of the nose, and in cases of syphilitic disease of the bones of the skull, there is a liability to cerebral abscess (Case 41).

Morbid Anatomy.—An abscess may form in any part of the brain. Usually it forms in the white substance, and when in the grey it is formed by extension from the white. The middle cerebral lobes are the most frequent seats of abscess. One hemisphere is as frequently attacked as the other. Of 80 cases, abscess was situated in the left hemisphere in 23, and in the right in 29. Practically, therefore, one hemisphere would appear to be as liable to be attacked as the other. In 12 cases abscess was situated in the middle lobe, but it is not stated in which hemisphere. The middle lobes were the seat of abscess in 23 out of 74 instances. Abscess was found in the cerebellum in 13 cases, in the pons Varolii twice, in the corpus striatum twice, in the optic thalamus twice. Abercrombie mentions an instance of abscess in the medulla oblongata. In several of the 74 cases the abscesses were multiple, and found in more than one part of the brain. The appearance of the abscess varies according to its duration. If it have been recently formed the pus is not inclosed in a cyst, but directly surrounded by ragged suppurating brain tissue, and there is not a trace of lining membrane to the cavity. If the abscess have been formed some time the pus is inclosed in a cyst of variable thickness. In very old abscesses the cyst wall has been found a quarter of an inch, or more, in thickness. When the abscess is a few weeks old the cyst wall is usually a line or two in thickness. The wall of the cyst is formed of fibro-cellular elements, and, in some cases, well-formed spindle-shaped fibres are seen; in others the fibro-cellular tissue has undergone granular degeneration, and the fibre cells are very indistinct. The cyst, when of old date, may be divided into three parts—an outer layer, which is made up of loose fine fibrous tissue; a middle layer, which is firmer and more coarsely fibrous than the outer; and the inner surface of the cyst is formed by a smooth, pyogenic membrane, in which some

record of such a case. Abscess is also said to have occurred when the carotid artery was tied. Probably it was softening of the brain, and not abscess.

small irregular dilated veins may be seen running in different directions.

In abscesses of recent formation, the pus is generally of a greenish hue, and may, or may not, have a disagreeable smell. In old abscesses the pus is green, fœtid, mucoid, and is decidedly alkaline. The pus removed from old abscesses, when placed under the microscope, shows few or no well-developed pus corpuscles; there is a large quantity of granular fat and granular matter without any nuclei.

There may be several encysted abscesses in the brain. In one of our cases there were no less than four; in another a large encysted abscess in each hemisphere.

The condition of the brain substance immediately around the abscess may vary very much; it has commonly undergone a process of softening. Rokitansky, speaking of recent abscess, says, round the abscess the brain substance is in a state of inflammation, producing red softening, yellow softening, and in more distant parts œdema of the brain tissue.

When a large abscess is situated in one of the hemispheres, the brain is often altered in shape; the convolutions being packed together and flattened; the hemisphere bulged at the side, and if the abscess be very large, the hemisphere containing it may feel more like a bag of pulpy thick fluid than solid brain substance. Collections of pus in the hemispheres tend to make their way towards, and discharge themselves into, the lateral ventricles, or on the surface of the brain. Pus, like blood, may fill one lateral ventricle only, or escape into the ventricle on the opposite side. In abscesses, as in very vascular, soft, gliomatous tumours of the brain, hæmorrhagic effusions are occasionally met with, and a coagulum of blood may be seen surrounded by pus.[1]

We have already stated that several abscesses may exist together in the brain; this is common when the patient has died of pyæmic cerebral abscess. In such cases every part of the brain may be studded with minute collections of pus; they may be found in the cerebrum, in the cerebellum, in the optic thalamus, in the corpus striatum, and pons Varolii. The size of these abscesses may vary from a pin's head to a hazel-nut, or even larger. They are usually situated near the surface of the brain. The cerebral substance around these pyæmic abscesses may be softened, at other times it is firm and comparatively healthy. When abscess of the brain is dependent upon disease of the internal ear, the morbid appearances are much as follows:—the dura mater, situated over the diseased petrous or mastoid portion of the temporal bone, is often found highly congested, softened, and ulcerated; or of a dirty green colour, and evidently sloughing, and the bone laid bare. In other cases the dura mater is simply thickened and covered with purulent lymph, and betwixt the dura mater and the bone there is often a collection of pus. The lateral sinuses are frequently involved and plugged, especially when there is disease of the mastoid cells; the sinus is often seen enveloped in pus and purulent lymph. The suppurative inflammation may extend along the internal jugular vein, and set up suppurative pleuritis and abscess in the lung.

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[1] See 'Guy's Hospital Reports,' vol. iii, 3rd series, Case No. 6, p. 291.

In abscess of the brain due to disease of the ear, there is, in the majority of cases, caries of the temporal bone; the latter is seen of a dark colour, with an irregular roughened surface. The abscess in the brain may have direct communication with the diseased bone, and the contents of the abscess make their way through the ulcerated openings in the dura and bone into the typanum, and then escape through the perforated membrane tympani into the external meatus, thus constituting what has been termed "otorrhœa cerebralis." A similar communication and escape of the pus is said to have occurred in cases of abscess in the brain caused by diseased ethmoid bone. At other times there is no such direct communication, for there is a layer of brain substance separating the abscess from the membrane of the brain. This layer is often softened, of an ash grey or yellowish appearance, and looking as if the pus was about to burst and discharge itself on the surface of the brain.

In some cases of abscess dependent on disease of the internal ear, there is no caries of the bone, as we have already mentioned; the membranes may be healthy, and the abscess may be situated at a distance greater or less from the surface of the brain.

Symptoms.—In 73 cases of abscess of the brain, the symptoms were as follows:—Pain in the head in 39 cases; epileptiform seizures in 38; coma in 30; heaviness, stupor, and drowsiness in 30; paralysis in 24; rigors in 17; pyrexia in 13; delirium in 13; vomiting in 12; incontinence of urine, or of fæces, or both, in 15; vertigo in 8; disordered sensibility, not including pain in the head, in 6; defective articulation in 4; defective sight in 3; an apoplectic attack in 1.

That some of the symptoms may have existed in greater proportion, we should be prepared to expect, especially such symptoms as vertigo, pyrexia, emaciation, and probably, in a greater number of cases, defect of sight would have been discovered had the eye been tested. The symptoms, therefore, that are most frequently observed in cases of abscess in the brain are pain in the head, epileptiform attacks, paralysis, coma, heaviness, drowsiness, stupor, rigors, pyrexia, delirium, vomiting, and incontinence of urine and fæces. In a few cases defective articulation was met with. The records show that the intellect was very little affected. Paralysis was observed in 24, that is in about one third, whereas in Lebert's cases it was observed in about one half. He included, however, not only local paralysis, but also general loss of muscular power, whereas we have confined the term to local paralysis only, such as loss of power on one side of the body, of one arm or leg, one side of the face, or some other part.

The first symptom, in many cases, is pain in the head; it may be the only indication of cerebral disease present for months. The pain is often very agonizing.[1]

An intense neuralgic pain situated over one spot is occasionally the first symptom; sometimes the pain is seated almost immediately over the region of the abscess. A boy having an abscess in the anterior lobe of the right hemisphere, complained of almost constant burning pain over the front and right side of the head, but this localisation of pain over the seat of the abscess is by no means constant. In some cases the pain is very remote. In one patient there was an abscess in the cerebellum, and the pain was felt in the forehead; in another there was an abscess in the right middle cerebral lobe, and the pain was referred to the left side of the head.

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[1] One patient lay in bed continuously holding his head with both his hands; another walked about with his hands pressed against one side of his head, crying out constantly, "Oh! my head; oh! my head." The pain is often so severe that the patients shriek from the agony they suffer. A patient, who was perfectly sensible, said he could not help screaming; and, although he tore and bit anybody or anything near him, he at the same time expressed contrition for what he was doing, and said the pain in his head was unbearable; it felt as if someone was knocking it with a hammer.

The pain often comes on in paroxysms; in other cases it is continuous, remittent, or intermittent. It is not present in all cases of cerebral abscess, as the statistics of our 76 cases show. It is very commonly associated with pain in the ear, when the abscess is due to disease of the auditory apparatus.

Instead of pain preceding, it may follow the convulsive attacks. Cases of this kind are by no means few.

Occasionally the first indication of cerebral mischief is a sudden and unexpected epileptiform seizure. The epileptiform seizures are occasionally the most prominent symptoms from the time of seizure to the patient's death. The epileptic attacks do not necessarily come on every day; occasionally some days elapse between the seizures.

After each convulsion the side affected is often left weak, and this increases until there is complete hemiplegia. The convulsive movements are sometimes unattended with insensibility, and are confined to one extremity, especially the arm. This had been long noticed.

Abercrombie alludes to a case of Lallemande's, in which there was pain in the right side of the head and tremor of the left arm. This was followed by continued convulsions, flexion, and extension of the left arm, which after some days ended in palsy.

Instead of convulsive movements, the first indications of brain disease may be numbness and tingling in one extremity.

The symptoms in other cases of cerebral abscess are like those that are said to indicate cerebral softening. There is sudden loss of power on one side of the body without any loss of consciousness; the leg being less affected than the arm.

In several instances rigors were very prominent symptoms throughout the attack. A patient, suffering from suppuration, was noticed to be getting thinner and weaker; when he was seized with rigors, diarrhœa, a dry brown parched tongue, and a hot skin, he became comatose and died. Pyæmic abscesses were discovered in the brain.

In some cases of pyæmic abscesses there are no special symptoms to show that organic disease is going on in the brain; but only the general indications of pyæmia. In others the accession of convulsive seizures, paralysis, or coma, indicates disease in the cerebral organ. Rigors so severe were noticed in a few instances, and returned with such regularity every day, that they closely resembled those of ague. One patient had headache, rigors, and vomiting, returning every day for five days, and then became unconscious. Rigors do not occur, in some instances, until after convulsive seizures have indicated cerebral mischief. Imperfect articulation, to a marked degree, was noticed in some cases, and in one there was loss of language.

With respect to the eye, Dr. Hughlings Jackson has mentioned to us that he has seen changes in the retina (optic neuritis ?) in a case of cerebral abscess. Dr. Jackson thinks such changes are common to several kinds of cerebral disease.

Mental disturbances were observed in some cases. Now and then the only symptoms noted were a heavy expression, a disinclination to speak, and indifference to surrounding objects. In some cases with disease of the ear, it was stated that the patients had attempted to commit suicide. One patient appeared to become hypochrondrical. Emaciation setting in rapidly was a marked symptom in several cases. Similar emaciation is seen in some cases of tumour of the brain; but is not so frequent as in abscess.

Patients suffering from cerebral abscess may have symptoms so closely resembling continued fever, that it is exceedingly difficult, if not impossible, with any degree of certainty, to say whether it be a case of fever or of organic disease of the brain.

PATHOLOGY.—Cerebral abscess may be produced by direct injury, or by *contre-coup;* contusing or lacerating the nervous tissue, and setting up inflammation and suppuration. It may be produced by suppurative inflammation in some tissue in the neighbourhood of the brain which spreads to a contiguous part; namely, in the ear or nose, which extends, and invades the dura mater, pia mater, and brain substance. Or the diseased action may spread by continuity of structure, as along a vein, and thus to the brain. Disease of the ear, nose, or of other cranial bones, may give rise to cerebral abscess in this manner. Again, abscess may be produced where there is disease of the cranial bones, or some growth involving them, by the veins communicating with the diseased bone becoming plugged. The process of coagulation extends and invades the veins communicating with the sinuses of the dura mater. These become plugged, as also the veins of the pia mater, and probably some branches entering the brain tissue also, and inflammation, terminating in suppuration, is thence set up in the brain. In other cases minute coagula, or thromboses, are supposed to be detached and carried along by the circulation until they are arrested in the capillaries of the brain, and often of the lungs, kidneys, and other organs.

Pyæmic abscesses are occasionally found in the brain, and not in any other organ of the body. Besides the coagula, some of the elements of pus may be carried by the circulation to aid in, or be the means of, setting up suppuration in the parts where the thrombosis is arrested. In this way abscesses in the brain are probably caused by abscess or suppuration in the liver, lungs, bowels, or in other parts.

We next enquire if every form of cerebral inflammation, or encephalitis, no matter what its origin, be liable to end in suppuration and an abscess. It has been many times stated that such is the case; but it would appear that the inflammation must be set up by a special cause, and unless it be so, it does not end in suppuration and abscess. Suppuration may apparently be excited by local injury or by the elements of pus or thrombosis; but experience shows that other forms of inflammation do not terminate in abscess. For instance, encephalitis and softening, the result of plugging of a cerebral artery, or encephalitis around a hæmorrhagic effusion, or around a gliomatous tumour or old cyst, shows no disposition to the formation of pus or abscess. The brain may soften, disintegrate, and a cyst may be formed, but there is no pus formed.

It is necessary, now, to ask if there be not good evidence to show that the brain may be the seat of suppurative inflammation and abscess without there being any cause to account for it? Is there not, in such cases, idiopathic inflammation which gives rise to idiopathic abscess? By idiopathic cerebral abscess, we suppose, is meant abscess which is not preceded or occasioned by injury or disease; its origin being unaccounted for. Lebert and others admit the occurrence of idiopathic cerebral abscess. Such cases are, however, in comparison with others, rare. It is beyond all doubt that a certain number of cases of cerebral abscess do occur in which no disease is discovered in any other part of the body, and there is no history of any recognised cause to account for the cerebral abscess.

Before, however, it be concluded that abscess has been formed idiopathically, it is necessary to remember that in the majority of cases there is a cause to account for the formation of such abscess, and that only in a very small minority have observers failed to find some admitted cause. In the face of such evidence, is there not good reason to think that in this small minority of cases, the primary cause has been overlooked? And, when it is still further remembered that hours have been passed in searching for the primary disease or cause, and at last it has been found limited to a mesenteric gland, a gumboil, or a whitlow—in fact the primary disease was so small, that it might have been very easily overlooked—it appears to us not difficult to understand how, even after very great care, the primary cause may have remained undiscovered. Bearing all this in mind, we recognise that in a few cases of cerebral abscess, the cause cannot be discovered; but even when the cause is undiscovered, we should not assume that the suppurative inflammation has commenced idiopathically in the brain.

Cerebral abscess proves fatal in many cases, not by a collection of pus in one or other part of the brain, but by extensive inflammatory softening around the abscess, involving vital parts of the brain; and it is from such softening that the abscess is able to make its way towards the ventricles or the surface of the brain. The softening around very old encysted abscess would appear not to be set up by pyogenic changes going on in the lining membrane, for there is not a large quantity of well-formed pus corpuscles in old encysted abscesses to show that such active changes have been going on in this membrane.

The softening would rather appear to be due to some circumstance interfering with the nutrition of the parts outside of the abscess, but in its neighbourhood. The nutrition of such parts, owing to the presence of a foreign body, being very feeble, it is easy to understand how a blow on the head or a debilitated or cachectic state of the system may be sufficient to excite such feebly nourished parts to take on acute inflammatory softening.

Has abscess in the brain any tendency to spontaneous cure? Lebert thinks not, and when we remember that there is no well-established case on record, showing that an abscess has been spontaneously cured, we readily admit that the evidence very strongly favours the belief that cerebral abscesses do not tend to a spontaneous cure. It is, however, necessary to remember that the brain is a very vital organ, severely taxed in our every-day labours, and, if not sound, its functions, which are essential to life, may be brought to a stop. When there is an abscess in the brain, the organ being unsound, its functions are very liable to be perverted, and death follows; whereas, if the abscess were seated in an organ less essential to life, any perversion of its functional activity would not be attended with fatal results, and thus time would be gained for the abscess to pass through the different stages essential for its cure. We may therefore ask ourselves whether it is that an abscess of the brain has no disposition to spontaneous cure, or whether it is that the patient does not live long enough for such a process to be accomplished? The development of a firm cyst wall would show that there is a disposition to spontaneous cure. The cyst wall is a protective effort that the brain makes to localise the mischief and protect the sound from the diseased part. And experience has shown that time is only required for such protecting efforts to be very great, and for the barrier guarding the pus to become stronger and stronger.

We are next led to ask, is there any thing in the condition of the pus discovered in old absceses to show that these were in a process of cure? To our minds, there is. It is usual to find such pus in a very degenerate condition, viz. granular and fatty, which is favourable to its absorption and concretion; such changes as occur in abscesses that have undergone spontaneous cure. This is no idle question. It is simply—Is cerebral abscess necessarily a fatal and incurable disease? Practically it is; but there is nothing in its morbid anatomy to lead us to conclude that it is necessarily incurable.

Diagnosis.—Cerebral abscess is inferred when there are symptoms of the brain indicative of organic disease, and there are present those morbid conditions that are known to give rise to cerebal abscess, such as a discharge from the ear, nose, or chronic suppuration elsewhere, or when there is a history of a blow, or of some other acknowledged cause of the disease. No doubt that in some cases the inference proves correct, where there is evidence showing that the cerebral substance is undoubtedly diseased, and further evidence of suppuration going on in some part of the body; for here there are indications of acute brain disease, and we are led to suspect that this is due to abscess, since such causes are present as are known to produce it. With the brain, however, as with other organs, we are more often able to say that it is diseased than to say what is the precise nature of the pathological changes going on in its substance.

There may be evidence to show that a patient has chronic disease of the nose or ear, and cerebral symptoms may supervene suddenly; epileptiform seizures and other symptoms may be present, such as are seen in cases of cerebral abscess; the patient may die, and yet there may be no disease of the brain or of its membranes. In some cases the membranes alone are diseased; in others the brain substance is softened without abscess. Disease of the bones of the skull, no matter whether it be fracture, syphilitic disease, or a growth, is liable to set up inflammation of the membranes of the brain, and the inflammation may spread and give rise to suppurative inflammation of the brain substance. If the patient survive six or seven weeks, an abscess may be formed; if he die in two or three weeks after acute symptoms have set in, the brain may be found softened, but without abscess. Not unfrequently death takes place before there is time for the suppurative inflammation to form an abscess.

There may be a history of injury to the head, cerebral disease may appear to have followed as a consequence, and the post-mortem examination reveal disease in the brain, but not abscess.

Injury may be followed by the formation, not of an abscess, but of a tumour, malignant disease, or by softening in the brain; or further, the disease may not be in the brain at all, but on the surface. Experience has shown that an injury to the head may produce a large cyst in the cavity of the arachnoid, and the symptoms of the case may be similar to what are seen in cases of encysted abscess.

A history of a blow on the head, followed by severe pain, loss of energy, altered manner, fits, and partial hemiplegia, occurs in abscess, but also in other cerebral diseases as well as abscess.

Cerebral symptoms, associated with offensive discharge from the ear and nose, would lead one to suspect abscess in the brain; but in one of our cases there was tumour, and not abscess. The co-existence of tumour in the brain, with the conditions that are known to produce abscess, makes the differential diagnosis extremely difficult. There are no pathognomic symptoms of abscess nor of tumour. It is only the different manner in which the symptoms are grouped, and the existence of those conditions that are known to produce one and not the other disease, which leads the practitioner to suspect that there may be tumour rather than abscess, or *vice versâ*.

The symptoms of abscess may differ from those of tumour in the following respects. In abscess there is often marked cachexia and great emaciation. In tumour the patients have often no marked cachexia, even look healthy, and the body is fairly nourished, certainly not emaciated. In abscess the duration of the cerebral symptoms is generally much shorter than in tumour. The symptoms in abscess are usually either latent or acute; in tumour they are often chronic. In the latter there may be local paralysis extending over several months, which is very rare in abscess. The intra-cranial nerves are much more frequently affected in tumour than in abscess. Occasionally, however, a person with tumour is seen to be much emaciated. These differences may enable the practitioner, in some cases, to diagnose one condition from the other, but in neither case are these differences so constant that a certain diagnosis can be made.

An abscess may lie latent in the brain for many months, and then acute symptoms may suddenly set in, and the patient die in a few days. The same thing may take place with respect to cerebral tumour. Experience has shown that cancerous deposits also may exist in the brain without there being any decided cerebral symptoms.

Chronic encysted abscesses and tumours of the brain have many symptoms in common. An hydatid tumour, gliomatous tumour, a cyst, cancerous deposits in the brain, or any other substance acting as a foreign body, may produce pain in the head, epileptiform seizures, with or without paralysis, optic neuritis, vomiting, or gradual loss of muscular power.

We are often able to say, when there is acute persistent but variable paralysis, with pyrexia, that there is acute inflammatory softening of the brain; but whether that softening is going on around an abscess, a tumour, or a cyst, or whether excited by disease situated on the surface of the brain, we may be unable to give any exact opinion.

With respect to rigors in cases of cerebral abscess, we have already stated that they are very well marked in some instances, and may be not unlike those of ague. This symptom is not, however, peculiar to cerebral abscess. It occasionally occurs in other forms of brain disease, for instance, as gliomatous tumours or tubercle.

Treatment of abscess of the brain should be by anticipation—obviating the causes which lead to it; in chronic disease of the ear or nose, by maintaining a free exit for the discharge, no matter what the exciting cause. Rest is the most important part of the treatment, avoiding thereby both mental and mechanical excitement.

By a simple diet and quiet life abscess may be dormant in the brain for an indefinite time.[1]

In cases were abscess follows injury to the head, surgical interference must be thought of. The principle in such cases is a mechanical one, namely, to reach the abscess and evacuate its contents, if that be thought advisable; experience has but little to commend it.

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[1] This is, however, to be observed that encysted abscess of the brain is fatal from changes outside the cyst of an acute kind, such as might be presumed to be preventible to a great extent. In support of this opinion we may say that, in our experience, we have known abscess lie quiet for months after a blow on the head, and the patient and the medical attendant become confident that all was well, the symptoms of lesion having slowly gone off; and yet a fatal issue be produced after a few hours' suffering by neglecting the precaution of rest and regimen. Probably such rest and care should be continued, not for months only, but for years. This we say from clinical observations of the changes in the cyst of old cerebral abscess.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
1	M.	21	Fracture of the skull from a blow of brickbat; no depression.	Fourteen days after the accident, on admission, he was almost insensible; stertorous breathing. Right forearm flexed and rigid. Incontinence of urine. Skull was trephined; a table-spoonful of pus escaped; he appeared to become more sensible. Paralysed on the right side. Skin hot. Pulse from 120 to 150. He became insensible, and died 9 days after the operation.	An abscess in the left hemisphere, situated immediately under the fractured portion of the skull.	16 days.	Mr. Maunder kindly supplied us with the particulars of this case. Admitted March 9th, 1864, into the London Hospital, under the care of Mr. Maunder.
2	M.	5	A blow on the forehead; skull fractured.	Skull was trephined, and he went out of hospital apparently well. In 2 months he was brought back with hemiplegia of the right side; he had had several convulsive seizures. 10 months after the injury he was again brought to the hospital; hemiplegia persisted, and subject to fits; optic neuritis. He died April 27th.	A large abscess occupying half the left hemisphere above the lateral ventricle. Very thick cyst.	Cannot be calculated.	This patient was under Mr. Maunder at the London Hospital.
3	M.	21	Disease of internal ear; caries of the temporal bone.	No history.	Pus in the arachnoid; in the right hemisphere a very large abscess, size and shape of a pear, opening into the lateral ventricle; brain around softened.	Ibid.	'St.Thom. Hosp. Post-mortem Records,' 1851. Dr. Bristowe has kindly allowed us to make use of these records.
4			Necrosis of the tibia.	Fell and hurt his leg a week before admission. Abscess formed; pyrexia set in, resembling typhoid; stupor; answered questions; tongue brown and dry; no rash. Diarrhœa the last few days.	Pyæmic abscess in the brain; three or four abscesses in the right cerebral hemisphere; abscesses in the lungs, spleen, and kidneys.	Ibid.	Ibid., 1858, p. 372.

No.	Sex.	Age	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
5			Fractured skull.	Not given.	In the left hemisphere a large encysted abscess; pyæmic deposits of the lung.	Ibid.	'St. Thom. Hosp. Post-mortem Records,' 1857.
6			Disease of the internal ear.		An encysted abscess in the left middle lobe.	Ibid.	Ibid.
7	M.	10	Injury to the head; in running struck his chin, and was thrown violently on the back of his head.	Same day as the injury a convulsive fit; left side chiefly affected; fits repeated. In the intervals of the fits, constant burning pain over the front and right side of his head, and conscious when spoken to. The last 24 hours passed his urine and fæces involuntarily. Skin cool; pulse 70. A succession of fits; coma.	Encysted abscess in the anterior lobe of the right hemisphere, size of a walnut; softening of the brain around; pus in the lateral ventricles.	Had occasional convulsive seizures 3 months and 3 days.	'St. Bart. Hosp. Post-mortem Records,' 1849. Dr. Andrew kindly allowed us to make use of these records.
8	F.	31	Abscess near the uterus.	Admitted a few hours before death, half unconscious. Very restless, lying on her back, and often tumbling out of bed. When roused could answer rationally. Not paralysed. Died suddenly.	Two or three collections of pus in the right hemisphere; two in the right lobe of the cerebellum, pus in the ventricles; abscesses in the liver; ulceration of the intestine, connected with an abscess near the uterus, which had opened into the intestine.	No history except of a few hours before death.	Ibid., vol. v, 1850.
9	M.	56	Acute pneumonia, right lung; grey hepatisation and purulent infiltration.	Supposed to have caught cold whilst riding outside an omnibus. Ill 3 weeks before admission. On admission into hospital, cough, fetid rusty expectoration; dulness and absence of respiratory sound over lower part of right lung. Much the same for 7 days, then appeared to improve during the next 18 days, and was able to get up; physical signs remained. 28th day.—Pain and swelling in the right leg, hardness in the course of the veins. Became worse, feverish, looked and felt ill; complexion sallow; delirium.	In the brain numerous collections of greenish, rather fetid pus—from a pin's head to a hazel-nut; one had opened into the right ventricle, one in the left lobe of the cerebellum; none in the optic thalamus, or corpus striatum, or pons.	Ill altogether about 2 months and 10 days. Brain symptoms about 10 days before he died.	Ibid., vol. v, 1850.

10	M.	30	Empyema of the right pleura; a cavity at the apex of the right lung; no tubercle.	Admitted 10 days before death in a state of great debility, with evidence of a vomica beneath the right clavicle; strength gradually failing. Cerebral substance around abscess firm; phlebitis in the right femoral vein. 2 days before death he was found unconscious, never roused, and gradually sank.	Right hemisphere of the brain bulged; in the posterior part of this hemisphere several large collections of greenish-yellow fetid pus, contained in cysts of concrete pus, size of a walnut to a pea; an abscess in the right optic thalamus; an abscess had burst into the lateral ventricles; brain substance around congested.	No brain symptoms until 2 days before death.	Ibid., vol. v, 1850.
11	F.	20	Old empyema in the left pleura; no tubercle.	Admitted 2 months before death with a copious, offensive, greenish, purulent expectoration. Physical signs.—Complete dulness all down left side behind; amphoric respiration and pectoriloquy. She improved, and able to walk about; physical signs remained the same. 3 weeks before her death she began to complain of headache; next day an epileptic seizure; 3 days afterwards another. Was pretty well for a week; then another fit, followed by as many as 18 in 24 hours, and was almost unconscious in the intervals. Following day became more conscious; fits returned, and recurred during the next 6 days; then she died.	Right hemisphere flattened; an encysted abscess in the middle of this hemisphere, and it had burst. Cyst walls firm, dense; a wineglassful of offensive pus, of a dirty greenish colour, streaked with brown. Brain substance soft and pulpy. A large suppurating cavity in the left pleura; no tubercle. Rest of the body not permitted to be examined.	3 weeks before death.	Ibid., vol. v, 1851.
12	M.		Unknown.	Admitted for dyspepsia, nausea, and sense of oppression; this continued for 14 days, then disappeared. About 3 months afterwards he was taciturn, heavy, and silent; answered questions rationally; urine passed involuntarily. 10 days afterwards pain in the crown of the head. 15 days after this he became comatose and died. No paralysis; sickness was the early and constant symptom.	Encysted abscess in both hemispheres, size of an orange; pus inodorous; brain substance around soft. Other organs healthy.	Acute symptoms began about 15 days before death.	'The Lancet,' July 12th, 1862, by Dr. Leith Adams.
13	M.	47	A fall on his head a few months before death.	Seized with convulsions, followed by hemiplegia; delirious. Sank into coma and died.	An abscess in the right hemisphere, opening into the right lateral ventricle.	Duration of acute symptoms, a week.	'Med. Times and Gaz.,' 1861, p. 196. Under the care of Dr. Tuke, York.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
14	M.	66	Discharge from the ear for several years.	Deafness on one side. Went to bed as well as usual; next morning paralysis one side of the face, also ptosis. Paralysis persisted for some days; became giddy; had severe rigors; drowsy; continually dozing unless spoken to; delirious at times; face flushed; head hot; tongue brown. He had an attack of convulsions. Gradually sank and died.	In the centre of the right cerebral hemisphere a large abscess.	Duration of acute symptoms, 23 days after admission into the hospital.	'Med. Times and Gaz.,' 1863. Under the care of Dr. Baly.
15	M.	16	Injury to the head; no fracture; bone laid bare.	Fell into a ship's hold. Had been subject to fits. Had one about half an hour after admission. For 9 days doing well, then drowsiness and stupor set in. Urine passed involuntarily; became greatly emaciated. Skull trephined; no relief. He died 47 days after the injury.	An abscess in the left anterior lobe contained an ounce of pus; rest of the brain healthy; no fracture. No other organ examined.	47 days.	Ibid., 1862, p. 267. Under the care of Dr. Lunn, Hull Infirmary.
16		18	Disease of the temporal bone.	None, except convulsions immediately before death.	A large cerebral abscess.	No acute symptoms until just before death.	Ibid., Dec., 1861. Under the care of Mons. Richel.
17			Scalp wound; bone dead.	Rapid emaciation. Heat of skin. Appetite was good. On the 28th day of admission allowed to get up; rigors in the night; unconscious next morning, but could be roused. Trephined; a little pus escaped. Died the 30th day.	Abscess in the left hemisphere, size of a hen's egg. No abscess elsewhere.	The rigors set in 2 days before death.	Ibid., Dec. 8th, 1860. Under the care of Mr. Paget.
18	M.	31	A nasal polypus by pressure obliterated the trunk of the internal carotid artery; absorption of the body of the sphenoid	Admitted for profuse epistaxis. A few days afterwards an epileptic seizure which left him hemiplegic on the left side. 10 days afterwards another fit. 13 hours afterwards coma and death.	Three abscesses in the right cerebral hemisphere; brain substance on that side much softer than on the opposite.	Some days; the number not given.	Ibid., June 19th, 1858. Under the care of Mr. Simon.

		dult	injury to the skull; no other internal abscess mentioned; no mention of the ear or nose.	headache, generally unwell. Had had pain in his head for 4 or 5 days; tongue brown and dry; thirsty; eyes suffused; became semi-comatose; pulse intermittent. Died 5 days afterwards. Body not emaciated.	A large abscess in the anterior part of the right hemisphere. Body not emaciated.	According to the Record, pain in the head about 19 days before death.	Ibid., Feb. 21st. Under Mr. Graut, 54th Regiment.
20	M.	34	Supposed cause, injury to the head.	Pain in the head and in his teeth and left side; tooth extracted. 15 days afterwards said to have had an attack of general convulsions; convulsions repeated; speech was slow; complexion became yellow; vomiting; facial paralysis on the right side. The patient died with signs of compression 5 months after the first appearance of symptoms.	Four encysted abscesses, two the size of a walnut, and two the size of a hazel-nut, in the left middle lobe; brain around the abscesses soft.	Doubtful.	'Archives générales de Médecine,' 1860, p. 672. Professor Greisinger, of Tübingen.
21	M.	20	Disease of the ear.	Had discharge from his ear nearly 4 years. On the 22nd December head and neck rigidly bent back, and spine curved; some rotatory movements of the head. On attempting to draw his head forward it elicited an expression of great pain. Was unable to swallow. Next day he suddenly became asphyxiated and died.	An abscess, the size of a walnut, in the pons Varolii, which had burst.		'Pathological Transactions,' vol. xi. Recorded by Dr. Down.
22	M.	22	Disease of the tympanum; no caries of the bone.	Sore throat for one week, and became generally ill. A discharge from the ear; redness and swelling in the throat; great depression. April 25th admitted, and May 2nd rigors; great prostration. 2 days afterwards pain in the right side; respiration quick. He became heavy and stupid, passed into a semi-comatose condition, and died.	An abscess, the size of a hen's egg, in the middle right lobe of the brain; abscess as large as a walnut in the right lobe of the cerebellum.		Ibid., vol. xv. Recorded by Dr. Dickinson. Patient under Dr. Barclay, St. George's Hospital.
23	F.	41	Suppuration of the right internal ear.	Discharge from the ear for several years. Admitted into St. Bartholomew's Hospital one month before death. Loss of power of right half of face; some spasmodic pain; constant pain right side of head; hyperæsthesia on the right. Became drowsy, semi-comatose, conscious when spoken to, and conversed with her friends the day before she died. No paralysis or irregular movements of the limbs. Could feed herself and stand up.	Abscess in the middle of the right lobe of the cerebellum; it communicated directly with the diseased portion of the temporal bone; the abscess encysted.	About 42 days.	'St. Bartholomew's Hospital Post-mortem Records,' vol. v.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records
24	M.	23	Caries of the temporal bone.	Admitted the day before his death, complaining of great pain in his head, especially in the back of his head; it felt like a coal of fire. Deaf on the right side, and troubled with earache since he had scarlet fever some years before. Occasionally vomited. Head hot. His illness commenced 11 days before admission, with rigor, followed by constant pain in his head. The night of admission pain agonising; he screamed with pain; quite conscious. Pain continued up to the time of his death. An hour before he died he became quiet. No paralysis; no irregular movements. He walked to the hospital the day before he died.	In the right lobe of the cerebellum one abscess the size of a walnut; the abscess had opened on the surface of the brain. Caries of the right temporal bone. Chronic ulcer of the stomach.	About 15 days.	'St. Bartholomew's Hospital Post-mortem Records,' vol. viii.
25	M.	25	Chronic disease of tympanum.	Two or three restless nights; severe frontal headache. On the 4th day vertigo and delirium, with slow pulse; efforts to vomit. 5th day cerebral oppression. 6th, paralytic weakness of the left side. 7th, coma; death.	Acute abscess in middle lobe of cerebrum on right side; dura mater sloughing; petrous bone carious.	About 7 days.	Recorded by Dr. Gull, 'Guy's Hosp. Reports,' vol. viii, 3rd ser.
26	F.	23	Chronic disease of tympanum.	Severe headache, principally over the right side of the head. Pain in the right ear, and frequent vomiting 14 days. Paroxysms of extreme restlessness; cerebral oppression. Death on the 17th day. No convulsion nor paralysis throughout.	Diffused suppuration and acute sloughing of the middle lobe of right hemisphere; dura mater over roof of tympanum sloughing; bone carious.	About 17 days.	Ibid.
27	F.	20	Disease of the tympanum following a blow on the ear.	Paralysis of right seventh nerve, and discharge from the ear, and headache after a blow. After 3 weeks headache increased, referred to forehead and occiput; pain on moving the neck; rigors, nausea, vomiting, sweating. No delirium. Death from syncope on the 4th day after the increase of the headache.	Abscess in cerebellum; inflammation of the vein of the aqueductus vestibuli.	About 12 days.	Ibid.

			disease of mucous membrane of nose.	interval, by convulsion and insensibility; recovery, and a second convulsion the same day. On the 3rd day headache, increasing to great intensity on the 5th; referred to the right side of the forehead, right temple, and occiput. No delirium. Death on the 8th day, in coma.	of cerebrum on the right side.		
29	M.	13	Chronic disease of the ear.	Languor for some days. Syncopal seizure; convulsions with insensibility, relieved by a discharge of pus from the right ear. Following day severe headache from the forehead to the vertex; nausea; delirium at night. 6th day, return of convulsions, with insensibility; intense pain and cramp in left leg. Death in rather sudden coma, 15th day.	Abscess, containing about an ounce and a half of pus under the posterior lobe of right hemisphere of the cerebrum, between it and the tentorium; caries of petrous bone.	15 days.	Ibid.
30	M.	25	Dysentery; chronic abscesses in liver; recent abscess in lung.	Epileptiform convulsion, followed by apoplectic symptoms lasting several hours. Only partial recovery of memory after 10 days. On the 14th vomiting; increased cerebral oppression. At beginning of 4th week sank into sudden coma, with paralysis of right side. Death on 26th day.	Undefined abscess in posterior lobe of cerebrum, on left side.	26 days.	Ibid.
31	M.	43	Cause not found.	Symptoms of hepatic derangement. During *apparent convalescence* sudden severe neuralgic pain over left eye; restlessness. On 3rd day defective articulation; delirium; gradually increasing coma. Death on 10th day.	Abscess, without definite walls, in anterior lobe of left hemisphere, near the surface; a second abscess, encysted, in the middle lobe on the right side; this had burst through the optic thalamus into the lateral ventricle.	10 days.	Ibid.
32	M.	46	Chronic abscess in sheath of left rectus abdominis muscle.	Under care for chronic abscess in abdominal walls. Rigor; drowsiness; great muscular debility, especially marked on the left side; partial paralysis of right side of face; difficult deglutition; semi-coma. Death on 6th day.	Five abscesses in different parts of the medullary substance of the right cerebral hemisphere; abscess in left hemisphere of cerebellum; abscesses not encysted.	6 days.	Ibid.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
33	M.	45	Suppuration of mesenteric glands after ileitis; abscess in spleen and kidney, not encysted.	Febrile symptoms; frequent rigors; pain, supposed to be neuralgic, in left side of head; sudden hemiplegia, without loss of consciousness; drowsiness. No delirium. Death by general exhaustion at end of 3 weeks.	Numerous abscesses, not encysted, scattered through the medullary substance of hemispheres; one in the right corpus striatum.	3 weeks.	Ibid.
34	M.	16	Disease in the orbit.	Phlegmonous inflammation of upper lip, extending to the right orbit. Abscess behind the globe relieved by puncture. Pain of an intermittent character over *left* side of the head, extending along the lower jaw and behind the ear. *Apparent convalescence.* Sudden dull, heavy pain in head, with vertigo. General convulsions, coma, and death, in about 5 hrs.	Abscess not encysted, occupying two thirds of the middle lobe of the brain on the right side; suppuration in orbit of the same side; sloughing dura mater.	Cannot be calculated.	Recorded by Dr. Gull.
35	M.	16	Fall on the back of the head.	Symptoms of typhus in a mild form. Headache, relieved by leeches. *Apparent convalescence.* Sudden insensibility, followed by a succession of epileptiform convulsions; partial recovery of consciousness; return of convulsions, followed by hemiplegia of right side. Death in coma after 10 weeks.	Large encysted abscess in middle lobe of cerebrum on the left side.	10 weeks.	Ibid.
36	F.	17	Cirrhosis of left lung, with large suppurating cavities (dilated bronchial tubes?).	During convalescence from variola, sudden maniacal delirium. Rigor; headache; drowsiness; paralysis of left arm. On 4th and 5th day severe pain in left leg. On 6th the leg was incessantly moved up and down in bed; sudden coma, and death about noon the same day.	An encysted abscess, which had burst through the right optic thalamus into the lateral ventricle; two smaller encysted abscesses in posterior third of left hemisphere.	8 days.	Ibid.
37	M.	34	Suppurating cavity in right lung during 3 years, after	Sudden seizure with vertigo, faintness, and loss of power on right side; no unconsciousness. Complete recovery, and good health for 8 months. Sudden clonic spasm of right arm, lasting a few minutes,	Large encysted abscess in posterior lobe of left cerebral hemisphere.	3 weeks from second seizure.	Ibid.

				beginning as before, but the attack soon became epileptiform. 4th day, return of epileptiform convulsions, followed by partial hemiplegia of right side, gradually becoming complete. Death after 3 weeks from second seizure.			
38	M.	23	Chronic disease of right pleura, with external fistulous opening; dilated bronchial tubes.	Sudden seizure, with only partial loss of consciousness. A second seizure the same day. After a month a third seizure, followed by impaired memory and general cerebral oppression. In the interval of the seizures, headache and occasional vomiting. Death at the end of 3 months.	Encysted abscess in posterior lobe of left cerebral hemisphere.	3 months.	Ibid.
39	M.	40	Fall on pavement; contrecoup, a year before distinct symptoms of cerebral disease.	Severe headache, principally frontal; gradual impairment of memory; transient attacks of loss of sight, sometimes with unconsciousness. These symptoms for a year. Subsequently, total blindness; continued headache; partial hemiplegia of left side. Frequent seizures, with general loss of muscular power; drowsiness; semi-coma. Death 14 months after the beginning of the symptoms.	Two large inflammatory cysts, with surrounding solid exudation in the anterior lobe of right cerebral hemisphere.	14 months from the commencement of chronic cerebral symptoms.	Ibid.
40	M.	42	Chronic disease of mucous membrane of nose.	Mucous discharges from nose for an uncertain period. Vertigo and headache 5 weeks. Seizure in bed at night; insensibility; paralysis of left arm and leg. Death after 12 days.	Softening and ulceration of the convolutions of the under surface of anterior lobe of right hemisphere, extending backwards to the fissure of Sylvius and inferior surface of corpus striatum.	12 days.	Ibid.
41	M.	35	Syphilitic disease of the bones of the head; caries of the cranium and perforation of the dura mater.	Had syphilis 5 years before. Was in the hospital 3 years before for diseased bones of the head; pieces of dead bone removed. 3 weeks before admission was delirious. On admission very weak; drowsy; vacant expression; fits of forgetfulness. Became more drowsy, and died the day after admission.	Anterior two thirds of the right cerebral hemisphere converted into a collection of fetid pus; entire brain somewhat softened.	About 3 weeks and 2 days.	Recorded by Dr. Ogle, 'British and Foreign Medico-Chirurgical Review,' No. lxx, p. 464.

Case.	Age.	Sex.	Symptoms.	Size of aneurism.	Authority.
9	41	M.	For some years sudden attacks of spasm of the throat, apparently brought on by hurry in swallowing food, or by cold externally. Splitting headache. Constipation. Three weeks before death vertigo and sudden insensibility, with stertor. Eyes open and fixed. Fæces passed involuntarily. On recovering consciousness partial paralysis of lower extremities, which are slightly flexed, and painful when extended. Another similar attack two days after, beginning with more severe spasm of throat. Continued tinuitus. Speech indistinct. Two other attacks, with more or less complete insensibility. Death sudden. After suddenly crying out "I am falling," he became insensible, and died in two minutes.	Effusion of serum under arachnoid and into ventricles. Aneurism of basilar size of pea. Old and recent extravasation of blood from rupture of sac. Arteries generally atheromatous.	Pfeufer, Henle and Pfeufer's 'Zeitschrift,' vol. i, p. 29.
10	20	F.	Dulness of apprehension. Deafness and tinnitus aurium. Undefined headache. A week before death excruciating headache; indistinctness of vision; pupil natural; vomiting and diarrhœa. Only slight disturbance of intellect. Death in an apoplectic stupor.	Aneurism, near division of basilar, size of bean. Extravasation into ventricles and about base of brain, from rupture of sac.	Hodgson, 'Diseased Arteries,' p. 76.
11	53	F.	Sudden and total deafness. General health unimpaired. After three years three seizures within two months, with loss of consciousness, stertor, and hemiplegia of left side. Able to walk, with assistance, after the attacks. Gradual loss of power for two years. Bedridden six weeks before death. One evening uttered a sudden scream; became unconscious; breathing slow; pupils contracted. Death the following day.	Large effusion of clear fluid into lateral ventricles. Aneurism size of hazelnut at middle of basilar. Extravasation of blood from rupture of the sac.	Dr. Van der Byl, 'Path. Trans.,' vol. vii, p. 122.
12	34	M.	After more or less continued headache for six months, convulsion in sleep, followed by cerebral oppression and deafness. Slowness and feebleness of all the voluntary movements, without distinct paralysis. Three weeks after, convulsion, sudden coma, and death in three days.	Aneurism size of large pea. Extravasation of blood from rupture of sac.	Author.

The symptoms of aneurism of the basilar artery, though not diagnostic of the nature of the particular lesion, form, upon the whole, a natural group indicating its presence and its seat. It is not so, however, where the middle cerebral artery is affected, for it will be seen that in such cases there was often no clinical history previous to the rupture of the sac; or, if any, none to indicate unequivocally the presence of organic lesion. Exceptions to this there are when the sac has become large, so as to compress the central parts about the base, as in the Case 4 below. Where the arteries of the circle of Willis are the seat of the aneurism, there may also be the same vagueness in the indications of organic disease; but in two cases where the posterior communicating artery was affected, ptosis, from compression of the third nerve, was an early symptom.

As with other tumours so with intracranial aneurism, headache, though difficult to estimate strictly, is one of the most important symptoms. The one character of it which should most arrest our attention is its constant recurrence and its often distressing severity, with concomitant disturbance of the cerebral functions. All care, however, will often fail to enable us to form a correct opinion; even should we, as some have suggested, auscultate the cranium for an aneurismal murmur!

Intracranial aneurism often serves to illustrate to us how much the whole nutrition of the brain may be affected by the operation of a strictly local lesion. It may lead to subarachnoid and ventricular effusion, and produce symptoms of insanity and epilepsy. In this the brain differs from the solid viscera of the abdomen. This is probably attributable to the quality of the normal action, the morbid condition being, in popular language, attributable to a disturbance of the nervous polarity. But whatever the explanation, this fact in the clinical history of cerebral lesions explains how various may be the phases of cases which, in their general anatomical details, may be alike, the degree to which this altered molecular change occurs varying, probably, according to the original quality of the nervous substance in different individuals.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
47	F.	44		Two weeks before admission much exposed to the sun's rays; felt giddy. Soon after fell into some water; much frightened. Manner became changed; miscalled objects; could not be understood. Difficulty in swallowing; no febrile symptoms; often raised her hand to her head. Complained of feeling ill; tongue furred; apathetic; right arm spasmodically flexed, and could not be straightened; right pupil dilated.	Membrane congested; anterior two thirds of the left cerebral hemisphere broken down into a mass of greenish purulent matter; in the middle lobe of the same a collection of yellow pus. Other organs presented nothing unusual. Pus in left ventricle.	Cannot be calculated.	Ibid. Case 92.
48	M.	44	Scalp wounds; arachnitis.	Pulled to the ground, and three or four scalp wounds produced over the right part of the forehead, penetrating to the bone. Went on well. In a day or two rigors came on; pulse 108; skin dry; tongue creamy; rigors returned; pain felt in the ankles and shoulders. He sank and died May 25th.	Outer surface of cranium roughened; pus between dura mater and bone, opposite one of the scalp wounds; pus in arachnoid; at one part dura mater ulcerated, and in the brain substance beneath was an abscess; the entire anterior lobe on the right side softened; superior longitudinal sinuses contained purulent fluid. Lungs, scrofulous deposits.	Died on the 18th day of admission.	Ibid. Case 89.
49	M.	25	Fracture of the occipital bone.	While drunk sustained a compound fracture of the occipital bone. Headache; restlessness; slight want of power in the muscles of the right side of the face; pupils dilated. Went on the same until 16th day, when seized with twitchings of the muscles of the face. Coma set in, and he died on the 16th day of admission.	Upper surface of both hemispheres smeared over with blood; white portion of the right anterior lobe bruised, and between it and the surface of the brain an abscess near the orbital plate of the ethmoid bone; pia mater thickened and congested; lateral ventricles distended with purulent fluid, and lining	15 days after admission. Headache came on 2 days after admission, and died 13 days afterwards.	Recorded by Dr. Ogle. Case 90.

50	M.	79	Carcinomatous ulceration of the integuments and bones of the face and walls of the right orbit.	During the whole of his disease there were no symptoms to indicate brain disease.	Ulceration and perforation of the sphenoid bone and dura mater; adjoining this near the convolutions an abscess in the substance of the right cerebral hemisphere, and it had contained a large quantity of pus which had escaped through the apertures in the dura mater and bone.	No symptoms.	Ibid. Case 76.
51	M.	25	Abscess in the liver communicating with the surface of the body.	Five weeks before admission had pain in the right side of abdomen; it abated and then recurred; was obliged to go to bed; hot skin; pain in the right side of belly, and some vomiting. Abdomen distended; walls hard and contracted. He much improved. Suddenly attacked with chills and heats. Got thinner and weaker. An abscess pointed and discharged at the umbilicus; pus became tinged with bile; pus in the stools. Rigors and diarrhœa came on; delirium; coma. Died in a typhoid state. No paralytic symptoms.	Numbers of minute abscesses in every part of the brain, in the white as well as in the grey matter, in the corpus striatum, optic thalamus, and pons Varolii, all smaller than a pea; surrounding brain softened. Secondary abscesses in the lungs. Several abscesses in the liver. Ulcers in the large intestine.	Cannot be estimated. No characteristic cerebral symptoms.	Ibid. Case 77.
52	M.	15	Superficial scalp wound of the forehead, caused by fall.	Drowsiness supervened, but he improved. 11 days after admission great headache; pulse quick. 14th day, two attacks of convulsions; delirium and pain came on; right pupil insensible to light; dilated. Later, passed evacuations involuntarily; spectral illusions; answered questions sensibly. Died on the 26th day after admission.	An abscess in the left cerebral hemisphere, anterior part; lateral ventricles full of pus; fracture of the left frontal bone; scalp was quite healed.	15 days before death.	Ibid. Case 78.
53	M.	25	A blow on the head; a scalp wound without fracture.	Blow left side of head. Severe headache 3 weeks afterwards, followed by rigors; sweating; articulation became affected, and slight hemiplegia on the right side supervened. Admitted into St. George's Hospital Nov. 24th, 1847. Bone of the scalp exposed. Symptoms relieved. Complete hemiplegia came on; skull trephined; foul pus escaped through a sloughy hole in the dura mater. Died 34 days after first symptoms set in.	No fracture of the skull; sloughy opening in the dura mater, leading to an abscess in the posterior part of the left cerebral hemisphere; walls firm and distinct; surrounding brain substance pulpy, and of a yellow colour. Other organs healthy. Lungs congested.	34 days.	Ibid. Case 79.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
54	M.	35	Had a cough for several years, and unable to work; but no proof of old-standing lung disease.	February 20th, numbness of the right arm and leg; gradually lost the power of that side. 24th, a fit; partially unconscious; hemiplegia became complete; much headache; mind unaffected; vomiting came on; great prostation. Gradually sank, and died 18 days after the numbness in the arm.	A circumscribed abscess, the size of an apricot, in the left cerebral hemisphere; middle lobe lined by a false membrane, contained dirty green pus; surrounding brain softened.	18 days.	Ibid. Case 80.
55	M.	26	No mention of abscess elsewhere; idiopathic?	On February 23rd, previously in good health, he was seized with hemiplegia of the left side; no loss of consciousness; sensibility of skin not much affected. On admission, left arm most paralysed; paralysis left side of the face; pain in the head. Under treatment he improved; recovered somewhat the use of his arm. 25 days after admission rigors set in—shook like a person in ague—followed by a fit and coma. Died next day.	Subarachnoid tissue infiltrated with pus; a circumscribed, encysted abscess above the roof of the right lateral ventricle; cyst wall was one twelfth of an inch thick; contained greenish fetid pus; surrounding brain softened; lateral ventricles filled with pus.	30 days.	Ibid. Case 81.
56	F.	53	Phagedænic wound following the removal of scirrhus of breast.	Admitted August 31st. September 8th she had an apoplectic attack, followed by coma; loss of power in the right arm and leg, and left side of face; difficult articulation. The arm partially recovered. Bedsores came on, and she died December 1st, that is, 53 days after the cerebral symptoms set in.	The left optic thalamus contained a quantity of purulent deposit, very much softened, to the extent of a threepenny piece, of an ochrey colour; left corpus striatum and neighbouring parts of the brain extensively softened. An abscess behind the cæcum; the latter ulcerated.	53 days.	Recorded by Dr. Ogle. Case 82.
57	M.	23	Abscess of the lung? Pyæmic abscess in the	Admitted March 13th, 1856. Been ill 3 weeks, with feverishness, shivering, and languor. On admission rapidly passed into a typhoid state; no rose-spots; had also pain in the head, and lost the	Several abscesses in the substance of the brain; two in the right hemisphere, about	30 days.	Ibid. Case 83.

				rationally; tongue dry and raw. Right arm became less paralysed. A succession of fits, and died March 22nd.	smaller ones. Abscesses in the left hippocampus major, left optic thalamus, and cerebellum. An abscess in the right lung.		
58	M.	35	Abscess following amputation of the arm; necrosis of the ulna; an abscess in the deltoid muscle.	After amputation of the forearm he left the hospital; got frequently drunk. He was readmitted October 31st, 1860; was shivering, sweating, and vomiting. The rigors recurred daily, almost at the same hour, reminding one of ague. An abscess formed in one of the buttocks. Cough, pain in the chest, bloody expectoration came on; surface became yellow. He died 27 days after admission.	A collection of purulent matter, the size of a filbert, was found in the lower part of the left middle lobe, not encysted. Secondary abscess in the lung. Other organs healthy.	Had symptoms 27 days before he died.	Ibid. Case 93.
59	M.	28	Disease of the ear.	Some years subject to occasional discharge from the left ear. 3 weeks before admission had a blow on the head from the edge of a door, followed by intense pain at the seat of the blow, and subsequently over the whole head. Week after delirious, and frequent attempts at self-destruction, and 25 days after became comatose and died.	A large abscess in the anterior and middle lobes of the left hemisphere, the walls of which were in a state of gangrene; abscess communicated with the tympanum by an ulcerated opening in the petrous portion of the temporal bone.	Symptoms set in 25 days before death.	Ibid. Case 97.
60	M.	27	Caries of the internal ear, and diseased lateral sinus.	Six months had purulent discharge from the right ear, deafness and pain in the head; it became fetid and copious. A month afterwards paralysis of the right side of the face, and tendency to stupor. Phthisical symptoms. Extensive hæmorrhage from the ear caused death.	Temporal bone carious; dura mater sloughing; under surface of the right middle cerebral lobe sloughy; medullary matter softened, and contained foul purulent fluid; lateral sinus connected with the carious temporal bone much inflamed, almost sloughy; foul pus in the cavity of tympanum. Scrofulous tubercles in the lungs and peritoneum. Bowels adherent.	Duration of symptoms 3 months and 9 days.	Ibid. Case 98.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
61	M.	8	Caries of the temporal bone.	Except slight discharge from the left ear since quite young, has been in good health up to six weeks ago, when he had a convulsive fit; it was preceded by vomiting. A second fit 7 days afterwards; left him with pain and discharge from his ear, and passing his motions involuntarily. Been "silly" since the fits. On admission paralysis of the left upper eyelid; mouth drawn to the left, and tongue turned to the right; limbs all weak, but perfect power over them; complained of nothing but of twinges of pain in his left ear and deafness; articulation imperfect. Became dull and drowsy; semi-comatose; then a convulsive attack; coma. Several fits followed. Coma, involuntary evacuations, and death.	Cerebral convolutions flattened; brain generally very vascular; in outer part of the left cerebral hemisphere, including the greater part of the middle lobe, a large abscess; walls firm, tough, and a quarter of an inch thick; lined with a blackish sloughy membrane, containing 6 oz. of fetid pus; surrounding brain soft and pulpy; it approached quite to the surface of the brain, and was adherent to the dura mater, covering the petrous portion of the temporal bone; at this spot a communication existed between the abscess and internal ear; ulcerated opening in the dura mater; cavity of the tympanum full of pus.	27 days before death.	Ibid. Case 99.
62	F.	26	Right temporal bone.	Six weeks before admission subject to boils. 2 weeks before admission pain in, and purulent discharge from, the right ear. 3 days before admission she became delirious. When admitted, great maniacal excitement; obliged to be confined in a strait jacket. 3 days afterwards slight attack of opisthotonos; retention of urine; incontinence of fæces. Sank into coma, and died.	Purulent fluid in the right arachnoid; convolutions generally flattened; brain throughout softened; in ventricles, 6 oz. of semi-purulent fluid; lining membrane vascular; septum lucidum quite destroyed; fornix diffluent; under surface of the middle	15 days before death.	Recorded by Dr. Ogle. Case 100.

					lined by a thick cyst of organized lymph, and containing 1 oz. of milk-white pus; fibrinous exudation between the surface and the bone; the latter slightly diseased and having an aperture leading to the tympanum, the latter ulcerated, full of pus.		
63	F.	51	Caries of temporal bone; coagulum in sinuses.	Admitted with cough, pain in limbs, neck, and throat, as if "a cold;" pulse quick, and a cachectic look; purulent discharge from the left ear. Recovered sufficiently to leave the hospital, but had slight headache and discharge from the ear. Next day had a fit, and day following insensible. Again admitted; incoherent, partially conscious; pulse full; tongue furred; very drowsy; urine passed involuntarily. Gradually sank, and died.	Dura mater ulcerated over a spot corresponding to a carious opening in the temporal bone; pia mater highly congested and ecchymosed; whole brain much softened, especially the left cerebral hemisphere, and in this a collection of pus, in a firm, distinct cyst; purulent fluid in the ventricle; septum broken down.	53 days before death.	Ibid. Case 101.
64	F.	23	Disease of the ear, following scarlet fever.	Subject to leucorrhœa; much headache for 2 weeks before admission. Had scarlet fever when a child; *seen double* ever since at times, and had discharge from the ear. Headache ever since a child. On admission, rambling; pulse quick and soft; never any rigors; thirst; a series of epileptic attacks; slight convulsion; strabismus; complained of great pain in the head, feeling "as if her eyes were shooting out;" much pain down the back and cardiac region; pyrexia; intense agony and screaming. Remained sensible until her death, which occurred without further supervention of convulsions.	Pus in the scalp over right temporal bone, with diploë full of pus; sloughy dura mater; pus between the latter and bone in the right, middle, and posterior fossæ; at this part, in the substance of the brain, an abscess, the size of a walnut; surrounding brain soft and vascular; left lateral sinus, coagulum of fibrin and blood.	29 days before death.	Ibid. Case 102.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
65	M.	54	Necrosis of the right temporal bone.	Six months before admission had a fit after a hearty meal, but no convulsive movement; soon recovered to some extent, but his mind remained affected, and he had attempted suicide. Frequent pains in the forehead, but no loss of muscular power. On admission, stupefied; soon became conscious; much pain in the head; purulent discharge from the right ear. 2 days after had a fit; loss of consciousness, but not convulsed; loss of sensibility; remained comatose; stertor, and died in a convulsive attack.	A very large abscess occupied the whole of the middle lobe of the right hemisphere down to the base, where the membranes were united together, and adherent to the petrous portion of the right temporal bone; lateral ventricle contained clear serum.	Cannot be calculated.	Ibid. Case 104.
66	F.	7	Disease of the left ear.	On admission discharge from the left ear; enlarged cervical glands and great debility. Scarlet fever 2 years ago. 4 months a fetid discharge from the left ear. On admission complete absence of fever; she much improved under treatment. One day, after syringing, she had a fit; she complained of pain in her head; she recognised people although the convulsive attacks persisted; never wandered in her mind. Fits continued, and she died.	Surface of the brain much flattened; much clear fluid in the lateral ventricles; septum lucidum softened; upper part of the left lobe of the cerebellum contained half an ounce of greenish pus in an irregular cavity; it communicated with the surface of the brain by an orifice corresponding to the internal auditory foramen of the left temporal bone, which was found to contain a quantity of pus; surface of the temporal bone natural; membrana tympani absent and bone of external auditory foramen exposed.	Undoubted cerebral symptoms 4 days before death.	Ibid. Case 105.
67	M.		Disease of the internal ear.	Admitted with sore throat, of a week's standing, and extreme difficulty in swallowing. Discharge from the right ear. *No ulceration* at the back of the throat. Offensive breath and foul tongue. Improved. Discharge from the ear ceased rather	Ulceration of the dura mater over the anterior surface of right temporal bone. Pus between the membrane and the bone. also in the lateral sinus and internal jugular vein.	Cannot be calculated.	Recorded by Dr. Ogle. Case 106.

				stupid, heavy, and comatose, and died.	the right middle lobe, the size of a hen's egg, full of pus. In the right lobe of the cerebellum was an abscess the size of a walnut, with an orifice, and attached to the dura mater. Pyæmic abscess in the lung. The temporal bone exposed on passing a probe into the external meatus.		
68	F.	26	Disease of the mucous membrane of the tympanum.	January 26th, 1846. Complained of earache on the right side. Had a discharge from the right ear since an attack of measles when a child; discharge very offensive at times. The last twelve or sixteen months had suffered from headaches, occasional forgetfulness, and giddiness. Pain increased, came on in paroxysms. February 17th. She was partially insensible, became comatose, and died twenty-three days after the pain commenced.	An abscess occupied the whole of the upper part of the right cerebral hemisphere. Surrounding brain healthy. Lymph on the dura mater covering petrous bone; membrane covering squamous bone was thick and detached. Tympanic mucous membrane, and that of the mastoid cells, were thick and soft, and covered with cheesy matter.	23 days.	Recorded by Mr. Toynbee, 'Diseases of the Ear,' p. 257, 1st edition.
69	F.	9½	Caries of the upper wall of tympanum; arachnitis.	Had measles when a child; offensive discharge from the ear, and occasional pain ever since. May 5th. Seized with symptoms of fever. Constant vomiting. On the 7th seemed well. 8th. The bad symptoms reappeared. 10th. Excruciating pain in the ear. Slight paralysis in the left side of face. Became comatose, and died twelve days after the appearance of first symptoms.	Pus in the left arachnoid. In the interior of the left middle cerebral lobe was an abscess the size of a hen's egg. Contained fetid pus. Caries in upper wall of tympanum. Dura mater ulcerated.	12 days.	Ibid., p. 259.
70	F.	12	Caries of the temporal bone; death following a blow on the head.	July 2nd, 1850. Received a violent blow on the head. 3rd. Had violent pain in the head, chiefly in the temporal region. Severe febrile symptoms. Symptoms increased. An abscess formed beneath the temporal muscle. Coma ensued. Death twenty-two days after the injury.	Dura mater lining the squamous bone thickened and adherent to the bone and arachnoid and a portion of the brain. An abscess in the middle lobe contained four ounces of pus. Petrous bone was diseased.	22 days.	Ibid., p. 308.

No.	Sex.	Age.	Cause.	Symptoms.	Seat of abscess.	How long before death acute symptoms set in.	Records.
71	M.	12	Caries of the right lateral sulcus; catarrhal inflammation of the muc. memb. lining the mastoid cells; retention of the discharge from the right ear.	Had scarlet fever two years ago. Discharge from the ear since. February 13th, 1854. Rigors and general malaise. Pyrexia. 15th. Abscess behind the ear. Slight stupor. Pain continued. 20th. He appeared a little better. 22nd. Decidedly improved. 23rd. The pain and feverishness returned. Drowsiness increased. Became weaker. Urine and fæces passed involuntarily. March 4th. Two severe rigors. Severe pain in the head. 6th. Pain increased. The following seven days suffered much less pain. 15th. Vomited much. 16th. Suddenly became convulsed and died.	An abscess was found in the middle lobe of cerebrum. Pus between the diseased mastoid process and dura mater which communicated with the superficial abscess behind the ear.	31 days.	Ibid., p. 322.
72	M.	35	Caries of the mastoid cells; polypi in the external meatus.	Suffered from frequent severe earache for some years, with discharge. Five weeks before death a polypus removed from the external meatus, soon followed by great pain at the back of the head, neck, and shoulder on the right side, supposed to be neuralgia. He became dull, heavy, and stupid, comatose, and died. This man's gait was very unsteady. He said people must have thought him intoxicated.	Abscess in the right lobe of cerebellum, containing fetid pus. Ulceration of dura mater over a carious portion of the temporal bone.	Cannot be calculated.	Ibid., p. 324.
73	M.	13	Caries of the petrous bone.	In June, 1851, suffered from so-called simple fever. He had severe headaches, chiefly in the right temporal region, and discharge from the right ear. Pain never entirely left. 12th. Pain increased. His speech became thick and indistinct, at times almost unintelligible. No fever. 13th. He dragged his right leg; vomited and became drowsy. 16th. Vomiting and drowsiness continued; had occasional double vision. 17th. Decidedly improved. 19th. Able to come downstairs. July 2nd. Symptoms returned with greater violence than ever; vomited frequently; intolerance of light; mind clear. July 7th. Again improved; pain, drowsiness, andsickness diminished. 13th. Much improved. 15th. Again he became	The under surface of the right lobe of cerebellum attached to the dura mater. Three abscesses in this lobe. Two lined by distinct membrane. Almost the whole of this lobe was a bank of pus.	Cannot be calculated accurately.	Ibid., p. 328.

74	M.	4	Injury to the head; a splinter of wood had penetrated the skull; this occurred on Oct. 1st, 1864. He died on Oct. 27th, 1864.	Suffered from the effects of the explosion at Erith, and received a penetrating wound of the skull by a piece of wood. A splinter had been driven in. It was removed, and, becoming worse, he was removed to Guy's Hospital. Mr. Cock made an incision. Some pus escaped, and after the pus some clear fluid followed. The child appeared relieved, and went to sleep; soon got worse, and died three days afterwards.	Autopsy showed an oval opening in the skull at the back part of the head on the right side. Acute arachnitis on both sides, more on the right. An abscess, the size of a walnut, in the back part of the right hemisphere, just below the descending cornua; purulent matter also in the lateral ventricle. The clear fluid that escaped was probably ventricular.
75	F.	31	Tubercular disease of the lungs, Fallopian tube, and uterus.	Two weeks before admission into Guy's Hospital she came one day as an out-patient, with a "wet rag" on the top of her head. She complained bitterly of the pain in her head. Her tongue was furred, and she had some pyrexia. Thinking it might be fever, she was ordered salines and advised to come into the hospital. About eleven days afterwards she came again, and "still had the rag on her head," and complained very much of pain in the head. This was her principal symptom.	Post-mortem examination showed no disease of the bones A small tumour attached to the surface of the dura mater, embedded into the edge of the hemisphere near the longitudinal sinus. Vascular arachnoid. On surface of brain a thick cyst the size of a grape, and filled with liquid pus. In the brain substance there were three abscesses exactly like it. Each had a thick cyst, which was vascular, and could be easily turned out. All close to the surface. All the size of small chestnuts. Lungs filled with tubercular matter. Fallopian tube near its end filled with a soft tubercular and purulent matter; also the uterus, near the opening of the tube.
76	M.	35	Cause unknown. Had he had any blow on the head? The so called fainting-fits evidence tended to show some long-standing cerebral affection?	Led an irregular life. Employed at a music hall, and some years ago got his chest crushed in an accident, and said after the injury that he spat blood. Not known that he had injured his head. All that could be learnt of any previous illness was that he had been subject to "fainting-fits." Said to have been well until three days before admission, when he was seized with tingling and numbness in the left hand. This increased; the arm got weak. On admission much loss of power on the left side; this increased. A week before he died, complete hemiplegia. Twitching on the right side. Became insensible and died.	*Post-mortem examination.*—Calvarium was irregularly thickened in parts, as if there might have been an osteitis at some previous time, but no caries was discovered. Arachnoid slightly greasy and opaque. A large abscess in the left hemisphere, with a distinct wall, evidently some weeks old. It contained offensive, viscid, yellow pus. Had opened into the left lateral ventricle. Lungs congested. Heart, liver, kidneys, bladder, spleen, peritoneum, all healthy. ⁂ The last three cases are recorded in 'Guy's Hospital Post-mortem Records.' Dr. Wilks has kindly allowed us to make use of them.

CASES

OF

ANEURISM OF THE CEREBRAL VESSELS.[1]

Aneurism of the cerebral vessels has been regarded as a disease of extreme rarity, and judging by the scanty records of it, we should conclude that the opinion was true. This apparent rarity, however, like all negative conclusions, is doubtful, and I think there is the more reason to suspect it as only apparent, and due to careless inquiry, since the discovery of these cases has been much more frequent during the last ten years. There are several reasons why intracranial aneurism is likely to be overlooked. First of all, as here hinted at, it has not been looked for, and it is notorious that the eye can see only that it brings with it the aptitude to see. Again, when death occurs from rupture of the sac, recent coagula may so imbed and conceal it that unless strictly looked for it will not be found, for the sac is often small and thin and transparent, except at the point of rupture. Further, also, when death has taken place from changes around the aneurism, as by pressure or softening, the sac itself may present such appearances that unless a minute dissection be made of it, its true nature may not be discovered. Whenever young persons die with symptoms of ingravescent apoplexy, and after death large effusion of blood is found, especially if the effusion be over the surface of the brain in the meshes of the pia mater, the presence of an aneurism is probable.

[1] Reprinted from the 'Guy's Hospital Reports,' vol. v, 1859, p. 281.

Though intracranial aneurism generally occurs on the larger trunks of the vessels as they lie at the base of the brain, or in the fissures between its lobes, the smaller branches, after entering the cerebral substance, are not exempt. Dr. Crisp records the case of a boy, aged fourteen, who died from rupture of one of two small aneurisms on the anterior cerebral artery in the substance of the anterior lobe. In the seventh volume of the 'Pathological Transactions' is a case by Dr. Van der Byl, where an aneurism on the posterior cerebral artery lay in the substance of the brain, as a tumour of the size of a hen's egg, composed of concentric layers of fibrin. In one of the cases given below (see Plate), it will be seen that death was occasioned by the rupture of a very small aneurism in the substance of the pons Varolii. This was found by hardening the brain-substance in spirit before removing the coagulum.

We are indebted to Dr. Brinton for a table of fifty-one cases[1] of intracranial aneurism, from which it appears that the most frequent seat of the disease is the basilar artery, and next the middle cerebral of either side. If to the cases in Dr. Brinton's table be added eleven others, four from the seventh volume of the 'Pathological Transactions,' and seven referred to in this paper, the results are as follows :

Seat of sixty-two cases of intracranial aneurism.

Vertebrals and branches	Vertebrals	4
	Basilar	20
	Small vessel in substance of pons	1
	Posterior cerebral	3
		28
Carotids and branches	Internal carotids by sella turcica	8
	Middle cerebral	15
	Anterior cerebral	6
	Anterior communicating	1
	Posterior communicating	4
		34

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[1] 'Transactions of Pathological Society,' vol. iii, p. 49. Note that in the table there are fifty-two cases, but No. 34 is omitted as not belonging to the category.

Of 58 of these cases, where the sex is given, 35 were males, and 23 females.

Men, it is well known, are more liable to all forms of aneurism than women, but there is great difference in the liability of the two sexes in respect to aneurism in different parts. Thus, in 137 cases of popliteal aneurism,[1] 133 were males, and only 4 females, or 33 to 1. Of 66 cases of aneurism of the femoral artery, 61 were males, 5 females, or 12 to 1. The difference lessens as we come to the aorta, where, of 167 cases of aneurism of the thoracic aorta, 132 were males, 35 females, or nearly 4 to 1. In carotid aneurism the liability of the two sexes appears to be nearly equal, for, of 25 cases, 13 were males, 12 females.

Fifty-eight cases, where the age is given, are distributed as follows:

Under 25 years	12
25 to 40 "	13
47 to 60 "	29
Over 60 "	4
	58

The relative importance of the disease at different ages is not, however, correctly expressed by these numbers, since in the later periods of life aneurism is not unfrequently found associated with more or less extensive disease of the cerebral vessels to which the symptoms and fatal results may be owing, the aneurism being an accidental and not important concomitant. In a case recorded by Dr. Bright (No. 5 in Dr. Brinton's table), in No. 25 of the same table, in Mr. Squire's case,[2] and also in one given below, the aneurismal dilatation of the middle cerebral, as it lay in the fissure of Sylvius, was unimportant, and in no way concerned in the fatal result, which was due in all these cases to atheromatous disease of the vessels generally, producing softening and effusion of blood. It is not so, however, in younger subjects. In them aneurism commonly occurs without disease of the vessels generally, and is fatal either from rupture of the sac or from pressure or softening around it. Of 20 cases occurring under thirty-five years of age, 16 were fatal by rupture of the sac, whilst of 37 cases over thirty-five, only 14 were fatal from rupture of the sac, or in other words, four cases out of five under thirty-five years of age were fatal directly from changes in the sac, but only two out of five over that age.

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[1] Crisp on 'Diseases of the Blood-vessels,' pp. 134, 224, 225.

[2] See Table, Case 56.

The youngest recorded age at which aneurism within the cranium has been fatal is fourteen years.

Aneurism of the intracranial arteries may exist as a general dilatation of the whole vessel for a more or less limited space. Mr. Hodgson[1] remarks, that the internal carotid not unfrequently exhibits this form of dilatation of its entire calibre immediately it enters the skull, and he also refers to a case where the vertebral arteries were in this way enlarged into two flask-like dilatations immediately before their junction to form the basilar. I am indebted to Dr. Brinton for the notes of a similar case, which occurred in the Royal Infirmary in Edinburgh. The patient, aged thirty-eight, was admitted with paralysis of both sides of the body, loss of speech and hearing, and yet seemingly conscious. On a post-mortem examination the basilar presented a fusiform dilatation the size of a goose-quill, which had produced softening of the pons to some depth.

The aneurism may be a simple pouch of all the coats, the pouched portion being as transparent and normal in appearance as the rest of the vessel, giving the impression that it might have been some original deformity. Usually intracranial aneurism is of this form, or, as surgeons term it, true aneurism, the walls of the aneurism subsequently undergoing gradual changes, partly from continued dilatation from within, partly from chronic inflammatory changes in the parts around, set up by the presence of the aneurism. There are four conditions under which aneurism of the intracranial arteries may exist: the sac may be a simple varicose dilatation, undergoing no further change, and not affecting the parts around; or it may slowly become obliterated by fibrinous coagula, and the continuity of the vessel be restored (such a case is recorded by Mr. Hodgson, in illustration of the cure of aneurism); or the sac may undergo slow distension, and at length rupture; or, lastly, it may act as a foreign body on the adjacent tissues, and set up inflammatory changes, or produce death by pressure.

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[1] Hodgson on 'Diseases of Arteries,' p. 76.

The conditions of the arterial supply to the brain are plainly such as reduce the pressure on the arteries which lie within the cranium to the lowest degree, and hence the tenuity of their coats. This tenuity renders them liable to aneurismal dilatation, from causes sometimes difficult to appreciate, but at others distinctly associated with mechanical injury to the cranium, or to violent muscular efforts. When we consider that this form of aneurism occurs in young persons, whose vessels are otherwise healthy, and that the most frequent seat of the dilatation is in those vessels which lie in contact with the bones at the base of the cranium, there is yet further probability that mechanical causes, acting locally, have much to do with the origin of disease.

There do not appear to be in the nature of the case any symptoms, or order of symptoms, upon which a diagnosis of cerebral aneurism can be made. The liability of the cerebral vessels to this lesion must, however, always enter into our calculations in the general diagnosis of tumours, especially when there is evidence of local pressure on parts about the base of the brain. From the frequency of basilar aneurism this would be most frequent on the pons Varolii, since there the conditions for pressure are most favourable. Symptoms may therefore, in such a case, begin from the earliest period of the formation of the aneurism, and continue for years before a fatal termination. It was so in Pfeufer's[1] case, where, according to the patient's account, he had for some years been subject to attacks of sudden inability to swallow. There was no regularity in the attacks, but they seem to be induced by swallowing food hurriedly, or by exposing the neck to cold, symptoms which indicated disturbance of the medulla oblongata, as it was afterwards proved, from the formation of an aneurism on the basilar artery. Subsequently he had headache, general convulsive attacks (ushered in by the convulsive affection of the throat), and paraplegic weakness of the lower extremities. In another case[1] sudden and absolute deafness was the earliest symptom of basilar aneurism, as long as four or five years before its fatal termination of rupture. The only pathological condition found was atrophy of each auditory nerve, which Dr. Van der Byl, who reports the case, thinks might have been produced by obstruction of the small branches of the basilar supplying the auditory nerves. The suggestion is important, as indicating how an aneurism may affect adjacent parts otherwise than by pressure. Deafness, as an early symptom of basilar aneurism, has been noticed in several other cases, and may have arisen from the cause here supposed, since the anatomical conditions about the sac itself did not seem sufficient to explain it. In contrast with these cases, which show how early the parts adjacent to an aneurism of the basilar may suffer, there are others where the sac has attained a much larger size without any symptoms to indicate its presence. Dr. Corfe, in his notes on the physiognomy of diseases[2] (Table, Case 3), relates the case of a man, about forty years of age, who was brought into the Middlesex Hospital in a state of insensibility into which he had fallen just before. The man had been working up to the time of the attack, and was apparently in the enjoyment of tolerable health. He died a few hours after admittance, and on a post-mortem examination there was found an aneurism of the basilar artery as large as an ordinary walnut, which covered the pons Varolii and a portion of the medulla oblongata. The same state of things occurred in a boy aged fourteen; and aneurism of the basilar as large as a walnut existed, without symptoms, until the system was disturbed by another cause of febrile excitement (Table, Case 4). The following table of cases of aneurism of the basilar artery exhibits a synopsis of the clinical history of the disease, and subsequently there is added a similar arrangement of clinical history of aneurism at other seats, from all which it will be confirmed that, although we may from the circumstances sometimes suspect the presence of aneurism within the cranium, we have, at the best, no symptoms upon which to ground more than a probable diagnosis.

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[1] 'Zeitschrift für Rationelle Medizin,' Erst Band, s. 293. (Case 9 in Synopsis.)

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[1] 'Pathological Transactions,' vol. vii, p. 123.

[2] 'Medical Times,' vol. xvi, p. 591.

Synopsis of Symptoms of Aneurism of the Basilar Artery.

Case.	Age.	Sex.	Symptoms.	Size of aneurism.	Authority.
1	35	M.	Gradually increasing impairment of muscular power throughout the body for two years before death. As symptoms advanced the tongue and lips were moved with difficulty; articulation indistinct; incontinence of urine, special senses and intellect unimpaired; fatuous expression of face; conjunctiva congested; pupils dilated; death preceded by symptoms of advancing lesion of the pons; general paralysis; loss of all power to articulate; peculiar tearful expression of features; laborious and slightly stertorous breathing.	Whole length of basilar artery dilated, forming a tumour as large as a chestnut, compressing the pons Varolii. The sac had not burst.	Dr. John Bright, 'Lancet,' 1828-9, p. 727.
2	54	M.	Headache, at times violent, yet able to continue work; great sense of weariness in limbs and sleepiness. Day preceding death was in usual health. Seized suddenly at night in sleep with sensation of being struck at the back of the neck; soon became comatose, with oppressed sobbing breathing, and paralysis of all the limbs. Partially recovered after bleeding, but died at 7 a.m. next morning.	Aneurism size of pea on basilar artery, fatal by rupture; blood effused over medulla oblongata and into spinal canal.	Mr. A. Jennings, 'Lancet,' 1832-3, p. 397.
3	35	M.	Frequent severe headache for a year; weight over temples; vertigo, chills, and rigors; difficulty in describing feelings; was thought childish, but continued to work as a tailor until five days before death, when he had a return of chilliness and rigors, with diplopia. After three days, paralysis of lower extremities, of right arm and right side of face, ptosis of right eye, and dilated pupil; sensation generally impaired, but chiefly on the right side; thick and indistinct articulation, as if fatuous; stupor, from which he could be roused, gradually passing into profound coma, with stertorous breathing. Death, on fifth day, from convulsion.	Small aneurism on basilar, half inch from origin; extravasation of blood at base, from rupture of sac.	Dr. Gordon, 'Medical Times,' vol. xix, p. 381.

4	40?	M.	Sudden seizure whilst at labour in apparent health; pupils contracted; respiration difficult; inability to swallow. Death after seven hours.	Aneurism of basilar, size of walnut; sac, containing soft coagulum, not ruptured.	Dr. Corfe, 'Medical Times,' vol. xvi, p. 591.
5	14	M.	For two years an aneurismal tumour of right internal carotid in neck. Except being unusually flurried when reproved, he ailed nothing; was strong, industrious, and intelligent, until sudden onset of fatal symptoms from cantharides, which produced purging, vomiting, and suppression of urine. Death on evening of second day, preceded by paralysis of right side, indistinct articulation, dysphagia, and laryngeal obstruction.	Aneurism of basilar artery, size of walnut; sac full of coagulum, not ruptured; large aneurism of internal carotid near its origin; other vessels healthy.	Dr. Kingston, 'Edinburgh Med. and Surg. Journ.,' vol. lvii, p. 69.
6	47	M.	Ill fourteen months. Entire hemiplegia of the left side, both of motion and sensation; the latter so complete, that an ignited body in contact with the skin is not felt; ptosis of left upper eyelid. Articulation and deglutition difficult. When sitting, needs support. Constant involuntary vibration to and fro of head and trunk and right foot. Right hand tremulous when used. Evacuations involuntary. Appetite voracious. Intellect unimpaired, but dull. When spoken to, expression cheerful. Occasional suicidal impulse. Mode of death not given.	Aneurism of basilar, size of pigeon's egg. Sac contained firm coagulum. Extravasation of blood from sac into softened substance of pons. Large effusion of limpid serum into meshes of pia mater and lateral ventricles.	W. Ruschenberger, 'American Journal of Med. Science,' vol. xii, p. 65.
7	58	M.	For nine months severe headache, not of a lancinating character, from occiput to frontal region, followed by paralysis of seventh nerve. A fortnight before death, during night, hemiplegia of left side, loss of speech, difficult deglutition. Intellect unimpaired. Died suddenly, whilst listening to a risible story.	Large effusion of serum into lateral ventricles. Aneurism of basilar near its division size of bean, comprising portio dura. Effusion of blood from rupture of sac.	Dr. Lager, 'Archiv. de Médecin,' vol. xii, p. 482.
8	59	M.	Long subject to indescribable sense of weight in head. Death from apoplexy following violent mental emotion.	Aneurism of basilar, near its division, size of pullet's egg. Large effusion of blood from rupture of sac.	M. Serres, 'Archiv. Générales de Médecine,' vol. x, p. 421.

Case.	Age.	Sex.	Symptoms.	Size of aneurism.	Authority.
9	41	M.	For some years sudden attacks of spasm of the throat, apparently brought on by hurry in swallowing food, or by cold externally. Splitting headache. Constipation. Three weeks before death vertigo and sudden insensibility, with stertor. Eyes open and fixed. Fæces passed involuntarily. On recovering consciousness partial paralysis of lower extremities, which are slightly flexed, and painful when extended. Another similar attack two days after, beginning with more severe spasm of throat. Continued tinnitus. Speech indistinct. Two other attacks, with more or less complete insensibility. Death sudden. After suddenly crying out "I am falling," he became insensible, and died in two minutes.	Effusion of serum under arachnoid and into ventricles. Aneurism of basilar size of pea. Old and recent extravasation of blood from rupture of sac. Arteries generally atheromatous.	Pfeufer, Henle and Pfeufer's 'Zeitschrift,' vol. i, p. 29.
10	20	F.	Dulness of apprehension. Deafness and tinnitus aurium. Undefined headache. A week before death excruciating headache; indistinctness of vision; pupil natural; vomiting and diarrhœa. Only slight disturbance of intellect. Death in an apoplectic stupor.	Aneurism, near division of basilar, size of bean. Extravasation into ventricles and about base of brain, from rupture of sac.	Hodgson, 'Diseased Arteries,' p. 76.
11	53	F.	Sudden and total deafness. General health unimpaired. After three years three seizures within two months, with loss of consciousness, stertor, and hemiplegia of left side. Able to walk, with assistance, after the attacks. Gradual loss of power for two years. Bedridden six weeks before death. One evening uttered a sudden scream; became unconscious; breathing slow; pupils contracted. Death the following day.	Large effusion of clear fluid into lateral ventricles. Aneurism size of hazelnut at middle of basilar. Extravasation of blood from rupture of the sac.	Dr. Van der Byl, 'Path. Trans.,' vol. vii, p. 122.
12	34	M.	After more or less continued headache for six months, convulsion in sleep, followed by cerebral oppression and deafness. Slowness and feebleness of all the voluntary movements, without distinct paralysis. Three weeks after, convulsion, sudden coma, and death in three days.	Aneurism size of large pea. Extravasation of blood from rupture of sac.	Author.

The symptoms of aneurism of the basilar artery, though not diagnostic of the nature of the particular lesion, form, upon the whole, a natural group indicating its presence and its seat. It is not so, however, where the middle cerebral artery is affected, for it will be seen that in such cases there was often no clinical history previous to the rupture of the sac; or, if any, none to indicate unequivocally the presence of organic lesion. Exceptions to this there are when the sac has become large, so as to compress the central parts about the base, as in the Case 4 below. Where the arteries of the circle of Willis are the seat of the aneurism, there may also be the same vagueness in the indications of organic disease; but in two cases where the posterior communicating artery was affected, ptosis, from compression of the third nerve, was an early symptom.

As with other tumours so with intracranial aneurism, headache, though difficult to estimate strictly, is one of the most important symptoms. The one character of it which should most arrest our attention is its constant recurrence and its often distressing severity, with concomitant disturbance of the cerebral functions. All care, however, will often fail to enable us to form a correct opinion; even should we, as some have suggested, auscultate the cranium for an aneurismal murmur!

Intracranial aneurism often serves to illustrate to us how much the whole nutrition of the brain may be affected by the operation of a strictly local lesion. It may lead to subarachnoid and ventricular effusion, and produce symptoms of insanity and epilepsy. In this the brain differs from the solid viscera of the abdomen. This is probably attributable to the quality of the normal action, the morbid condition being, in popular language, attributable to a disturbance of the nervous polarity. But whatever the explanation, this fact in the clinical history of cerebral lesions explains how various may be the phases of cases which, in their general anatomical details, may be alike, the degree to which this altered molecular change occurs varying, probably, according to the original quality of the nervous substance in different individuals.

Synopsis of Symptoms of Aneurism of Middle Cerebral Artery.

Case.	Age.	Sex.	Symptoms.	Size of aneurism.	Authority.
1	35	M.	No history of previous cerebral symptoms. Under treatment for hæmatemesis. Whilst walking across ward suddenly cried out from pain in the head, and fell down insensible. Breathing slow and interrupted. Death in thirty minutes.	Large extravasation of blood from rupture of aneurism, size of a swan-shot, in branch of middle cerebral artery.	'Med. Gazette,' vol. xxiii, p. 453.
2	19	M.	No history of previous cerebral symptoms. Sudden exclamation, "Oh, my head," followed by insensibility. Recovery of consciousness after some hours; a gradual improvement during a week. Relapse into insensibility, and death in six hours.	Aneurism of middle cerebral, near origin. Effusion of blood to a large extent over left hemisphere from rupture of sac.	'Bright's Med. Reports,' vol. ii, p. 266.
3	65	F.	Sixteen days before death sudden apoplectic seizure without premonitory symptoms. Health good at the time. Complete hemiplegia of right side and general lethargy. General improvement during a fortnight, followed by relapse into apoplectic state, and death after a few hours.	Large effusion of blood under arachnoid, over surface of left hemisphere, and breaking through into lateral ventricles, from rupture of an aneurism of middle cerebral artery. Sac imbedded in cerebral substance. Other cerebral trunks healthy.	'Medico-Chir. Review,' vol. xiv, p. 234.
4	21	F.	Fifteen months before death frontal headache, gradually increasing in violence, and followed by seizure regarded as hysterical, in which there was insensibility for two hours. Recovery. After a month another similar seizure. From that time drowsiness and continued and distressing headache, loss of strength, and emaciation; appetite continuing good. At the end of a year loss of sight of right eye, with dilated pupil. Face drawn to right side when smiling. Memory defective on recent events. Died in coma, with stertorous breathing.	Four ounces of serous blood in ventricles. Aneurism size of hen's egg, resting on right half of sella turcica, compressing anterior and middle lobes of the brain, the optic and olfactory nerves, and crus cerebri, and producing absorption of the sphenoid bone.	Dr. Hamilton Roe, 'Path. Soc. Transact.,' vol. iii, p. 46.

				tion of blood into ventricles.	vol. vii, p. 127.
6	17	F.	Severe headache, continuing three days, followed by hemiplegia and coma. Gradual exhaustion. Sudden exacerbation of symptoms a few minutes before death, eight weeks from onset of symptoms.	Aneurism of left middle cerebral artery in the anterior part of left middle lobe of cerebrum. Softening around the sac. Laceration of corpus striatum and thalamus opticus by large effusion of blood, from bursting of the sac. Lateral ventricles full of blood.	Author.

Case of Aneurism of Anterior Cerebral Artery in substance of Hemisphere.

	14	M.	Whilst carrying heavy weight was seized with giddiness, and became insensible for ten minutes. On recovery, had feeling of weight in the head, continued vertigo, and intolerance of light. After seventeen days suddenly fell, and became comatose; breathing stertorous; pupils dilated; paralysis of left side. Died on the fifth day.	Two aneurisms, one size of pea, other of horse-bean, on anterior cerebral artery, in substance of hemisphere. Rupture of larger sac and effusion of blood, breaking into right lateral ventricle and through septum lucidum into left. Smaller sac full of laminated fibrin.	Dr. Crisp, 'Diseases of Arteries,' p. 165.

Synopsis of Symptoms of Aneurism of the Arteries of the Circle of Willis.

1	62	F.	No symptoms from the presence of the aneurism. Patient retired to bed in usual health. Sudden seizure in the morning on rising; semi-convulsions, passing rapidly into coma. Death in less than an hour.	Aneurism, size of filbert, arising from junction of posterior communicating and carotid arteries. Sac ruptured. Hæmorrhage into ventricles and about base.	Dr. Francis, 'Med. Gaz.,' vol. xxxviii, p. 213.
2	54	M.	Insane for three years. Occasional paroxysms of violent excitement. Six months before death epileptiform seizures. Impairment of hearing. No paralysis. Sudden prostration, with slow but not stertorous breathing. Semi-coma.	Brain small. Multilocular aneurism, size of small apple, on posterior communicating artery, occupying floor of third ventricle, destroying the tuber cinereum, the origin of the optic and the olfactory on the same side. Optic nerve of opposite side flattened and softened.	Mr. R. Smith, 'Dublin Journal,' vol. xxv, p. 507.

Case.	Age.	Sex.	Symptoms.	Size of aneurism.	Authority.
3	18	F.	Severe headache and symptoms of acute mania two years before death. Recovery after several months, but continued severe and "heavy" pains in the head, with occasional vomiting. Ptosis of left eyelid, gradually increasing for a week; globe on same side abducted and fixed outwards; pupil large. Shooting pain in left temple, relieved by pressure. Sudden seizure without convulsion. Coma. Death in ten minutes.	Effusion of blood under arachnoid from optic commissure backwards over pons Varolii and medulla oblongata. Clot in fourth ventricle. Aneurism on left posterior communicating artery size of filbert. Rupture of sac.	Dr. Hare, 'London Journal of Medicine,' 1850, p. 824.
4	30	M.	Working, in apparent health, and drinking freely. Sudden pain in head, presently followed by coma, difficult and stertorous breathing, foaming at mouth. Contracted, fixed pupils. Arms extended, and at times slightly convulsed. Death, in continued coma, after four or five hours.	Effusion of blood over surface of brain and into third and fourth ventricles, from rupture of aneurism on right anterior cerebral artery. The under part of right hemisphere and floor of third ventricle torn by the effusion.	J. King, 'Med. Quarterly Review,' 1834, 1835, p. 434.
5	20	F.	Headache and vertigo three weeks. Sudden pain over right eye; faintness and vomiting. Next day ptosis of right eyelid, and impaired vision on same side; eye abducted; pupil dilated. No attendant pain and vertigo. After another three weeks found in bed in the morning insensible; breathing stertorous. Died in four hours.	Aneurism, size of pea, on posterior communicating artery, containing solid fibrinous coagulum. This had compressed and flattened third nerve. Recent hæmorrhage from rupture of sac.	Mr. France, 'Guy's Hospital Reports,' vol. iv, p. 46.
6	35	M.	For five years, at intervals, headache, vertigo, and tinnitus. Headache sometimes severe, commonly referred to the occiput. Sudden seizure, with vomiting and convulsions, soon passing into profound coma. Death in eight hours.	Surface of brain deluged with blood effused under the arachnoid from rupture of aneurism on anterior cerebral artery of left side. Floor of third ventricle lacerated. Ventricles full of blood.	Author.

NOTE.—A tabulated statement of 86 cases, published subsequently to the above, is given by Dr. Peacock in a paper on "Intra-cranial Aneurisms" in the 'St Thomas's Hospital Reports' for 1876.—ED.

Case i.—*After more or less continued headache for six months, a convulsion in sleep, followed by cerebral oppression and deafness; slowness and feebleness of all the voluntary movements, without distinct paralysis; about three weeks after seizure sudden coma and death in three days; aneurism of the basilar artery; superficial softening of pons Varolii; effusion of blood from rupture of the sac.*

Hugh B—, æt. 34, a large, tall, heavy-looking man, with a syphilitic blotch on the upper lip, was admitted under my care into Guy's Hospital, December 24th, 1855. In 1853 he had a severe blow on the back of the head when drunk, and was laid up for a fortnight. After this he frequently vomited in the morning, which was at the time attributed to his drunken habits. In the summer of 1855 he began to complain of headache, but continued working in his usual health until a fortnight before his admission into the hospital. At this time he had a convulsion in his sleep, and remained insensible for three days. On recovering his consciousness, the mind was dull and the memory very defective. When first visited, the following note was made of his condition:—"He has an anxious oppressed look. After repeated questioning to bring him to the subject, he gives a generally correct account of himself, but his memory is obviously defective. He has pain at the back of the head and down the neck, and cannot bend the head forward with freedom. Pressure over the transverse processes on the right side of the neck, and deep down to the occipital condyle, makes him complain. When asked if he has headache, he says, slowly and with indifference, 'Yes, occasionally.' He often puts his hand to his head and exclaims, 'Oh, dear.' There is a marked degree of deafness on the right side, which dates from the seizure. There is no paralysis of the extremities, or of the muscles of expression, but all the voluntary movements are performed slowly and feebly. Urine and fæces sometimes passed involuntarily. No difficulty in deglutition. No vomiting or nausea. Pupils rather large and sluggish; the left oval, and somewhat larger than the right. Sight unaffected. Pulse 60. Rhythm regular. Respiration 20, with occasional slight and ineffectual cough, as if restrained by fear of shaking the head. He was emaciated considerably since the seizure. Abdomen sunken. Constipation. Urine abundant, without deposits; not albuminous. Surface cool. Extremities cold. Mouth affected by mercury." After this report there was no important change in his symptoms until January 4th. His appetite was rather voracious. He continued to complain of pain in the back of the head and in the neck when he was moved. The whole muscular system became much enfeebled, but without distinct paralysis of any part. Early in the morning of the 4th he was found comatose, with dilated pupils, eyes in constant oscillating movement, and slight convergent strabismus. Urine and fæces passed under him. Face inexpressive. Breathing stertorous. Pulse 76. Respirations 17. Conjunctivæ injected; the right covered with muco-purulent secretion. On the 5th his state was the same. On the 6th the skin was very hot and sweating. Respirations 40, stertorous. Pulse 160. Death early on the morning of the 7th, one month from the first seizure.

Post-mortem examination.—Effusion of clear fluid into ventricles. At the anterior part of the basilar artery there was an aneurism the size of a small nut. The sac contained a firm clot. Around it, into the subarachnoid space there was recently effused blood. Fibrinous coagulum, continuous with that in the sac, extended into the trunk of the artery in both directions. The superior cerebellar and the posterior cerebral arteries were pervious. The posterior communicating arteries were filled with recent coagulum. The substance of the pons beneath the aneurism seemed to the eye quite uninjured, but a microscopical examination detected numerous "granule-masses" in the superficial parts. The blood-vessels in all the other parts of the brain were healthy. The effusion of blood around the sac was evidently as recent as the last seizure, three days before death. The cervical spine was carefully examined; the cord, nerves, and all the other textures were healthy. The thoracic and abdominal viscera were healthy.

In this case and in Case 6 there was a distinct history of injury, and in both the arteries in the other parts beyond the aneurism were perfectly healthy. The symptoms were plainly indicative of disease at the base of the brain, and a consideration of the whole clinical history of basilar aneurism should in a similar case suggest the probable nature of the lesion. The inflammatory changes around the sac were slowly bringing matters to a fatal issue, apart from rupture of it; but as they advanced the sac received less support from the softened nerve-substance, and hæmorrhage kindly cut the thread of life.

Case 2.—*Aneurism in the substance of the pons Varolii; ingravescent apoplexy; death in three hours and a half.*

Mrs. W—, æt. 43, wife of a publican, complained for about a fortnight before her death of dyspepsia, flatulence, and headache, which she described as at times "quite overpowering." At 7 p.m., February 26th, 1858, she was assisting at the bar, when suddenly she cried out, "Oh, my head—I'm dying!" and fell backwards. She never spoke afterwards, but was partially conscious for two hours, being able to open her mouth when told to do so, and to move the left leg and arm. When visited at half past 9 p.m., she was perfectly comatose; lay supine, with the limbs extended and flaccid. The jaw fallen. The pupils minutely contracted and immoveable. Breathing greatly embarrassed from paralysis of the larynx. Respirations 36, stertorous, and with an expiratory moan. Walls of chest scarcely moving during inspiration, the larynx at the same time descending, and the supra-clavicular spaces being depressed; now and then a fuller sighing inspiration. Pulse varying from 70 to 90 in the minute; irregular in force and rhythm. Carotids throbbing. Face pale. Muscles of deglutition and tongue quite paralysed. Occasional ineffectual efforts to vomit, with the expulsion of a little mucus. There had been no involuntary evacuation of the rectum or bladder. Urine drawn off by the catheter pale, and containing a small quantity of albumen. Death the same evening, three hours and a half from the seizure.

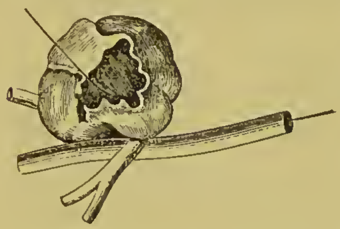
Post-mortem examination.—Arcus senilis well marked. A large quantity of subcutaneous fat on the abdomen. Heart normal in size and structure. Kidneys under the average size; their surface granular, and tunics adherent. Arachnoid of the surface of the brain granular and opalescent, with several drachms of clear fluid in the meshes of the pia mater. Lateral ventricles healthy. In the lower third of the pons Varolii, in the middle line, a recent coagulum weighing two drachms. On removing this a pyriform aneurism, having much the appearance and size of a withered grain of wheat, was seen projecting from the floor of the cavity produced by the effusion of blood. The blood had escaped from a longitudinal slit in the sac. The brain-substance around the coagulum presented no evidence of softening preceding the effusion of blood. The basilar artery was mottled throughout by opaque fatty deposits.

The seat and minute size of the aneurism in this case are the chief points of interest in it. It presents in other particulars the ordinary history of apoplexy of the pons Varolii. It is a question raised by this case how far atheromatous changes in the larger arteries may throw the ventricular impulse upon the smaller vessels of the brain, and lead to aneurismal dilatation of them. The flatulence and dyspepsia which for a fortnight preceded the apoplectic seizure may have been premonitory of it. The more decided disturbance of the stomach in nausea and vomiting is notoriously often referable to the brain, and there is reason to believe that the pneumogastric nerves are frequently the channels through which early symptoms of impending apoplexy exhibit themselves.

Case 3.—*Severe headache, continuing three days, followed by hemiplegia and coma; gradual exhaustion; sudden exacerbation of symptoms a few minutes before death, eight weeks from onset of symptoms; aneurism of left middle cerebral artery in the anterior part of left middle lobe of cerebrum; softening around the sac; laceration of corpus striatum and thalamus opticus by large effusion of blood from bursting of the sac; lateral ventricles full of blood.*

Louisa B—, æt. 17, a fair, delicate girl, employed as a domestic servant, enjoyed good health until two months before her death. About this period she became the subject of rheumatism from exposure to cold and damp. Her rheumatic symptoms amounted only to slight swelling of the right knee, and wandering pains in the limbs; she was still able to continue her work. January 1st, 1858, she complained of headache, which was unusually severe on the 3rd, and in the afternoon of that day, whilst talking to a friend, she suddenly lost the power of speech, and became paralysed on the right side. There was no reliable account of her condition from this date until the 15th, when she was admitted into Guy's Hospital, under the care of my colleague, Dr. Owen Rees. Her skin was then hot and dry. She lay comatose, with the right side paralysed. Pupils dilated. Urine and fæces passed unconsciously. Bedsore over sacrum. Heart's action sharp. Pulse 110. During ten days after her admission there was no important change except increased exhaustion. She frequently moved the left hand to her head, as if in pain. On the 25th the pulse was not perceptible at the wrist. On the 26th, at half-past 11 a.m., she suddenly gave a scream, and the face became congested. Death after a few minutes, without convulsions.

Post-mortem examination.—In the substance of the middle lobe of the cerebrum on the left side, and on the principal division of the middle meningeal artery, there was an aneurism of the size of a small nut, surrounded by a large recent coagulum and softened brain-tissue. Part of the coagulum consisted of fibrin which had separated from the effused blood, as in the formation of the buffy coat. The anterior third of the corpus striatum and the principal part of the thalamus opticus was broken up by the effusion, which had also filled the lateral ventricles and surrounded the crura cerebri and medulla oblongata. The arteries were healthy; weight of heart nine ounces. Valves healthy, with the exception of some granulations on the mitral. Spleen large, full of blood, and containing several white fibrinous masses; a section of one of the largest of these presented numerous points of softening. Kidneys large, with similar fibrinous masses (embolic), with blood effused around them. The outer coat of the aneurism consisted of a very thin layer of areolar tissue, the thicker part of the sac of laminated toughish fibrin.



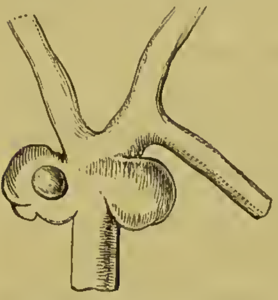
Remarks.—The previous occurrence of rheumatism, with signs of endocarditis, led at first to the opinion that the hemiplegia might be owing to emboli obstructing one of the cerebral vessels. This was apparently confirmed at the post-mortem examination by the fibrinous exudations in the liver and spleen, and it was only after careful search that the aneurismal sac was discovered in the midst of the softened tissue and clot. The effusion of blood had evidently taken place at the time of the last and fatal seizure. There was no trace of any old coagulum, so that we must refer the sudden hemiplegia to ramollissement only, around the sac. These changes were gradually inducing exhaustion when, as usual, the sac ruptured and cut short the case by profuse hæmorrhage. What determined the presence and seat of the aneurism could not be conjectured. The arteries elsewhere were entirely free from disease, and so was the trunk of the vessel in which the aneurism was seated.

Case 4.—*Ingravescent apoplexy from rupture of an aneurism on middle cerebral artery of left side; death on the sixth day.*

Fanny S—, æt. 30, of middle stature, dark complexion, of rather emaciated and cachectic aspect, was admitted into Guy's Hospital on the 5th of November, 1850. The account given was, that she was cook in a family, and the previous evening had left home in perfect health, accompanied by a female friend. Whilst walking she suddenly called out, "Oh, my head!" and put up her left hand. She vomited, and, as her friend thought, fainted. After a brief interval she partially recovered, and was able to walk back to her residence with the support of two men. By the time she reached home she had recovered her consciousness sufficiently to ask her friend to conceal the fact of her having left the house. She took some tea and walked upstairs to bed, and was left, as it was supposed, asleep, but in reality in a state of gradually increasing coma. When admitted into the hospital at noon the following day only a slight impression could be made by any attempt to rouse her. The right arm was quite paralysed, the muscles flaccid; the right leg in the same condition, with only slight traces of excito-motor action when the sole of the foot was tickled. Features inexpressive. Both pupils contracted. Respirations 32, tranquil. Pulse 70. Heart's action sharp, without any abnormal murmur. Urine drawn off by the catheter free from albumen. In the evening the left pupil had dilated, and was immovable on the stimulus of light; the right remained contracted. The left eyelid was slightly fallen. The patient turned herself over in bed in a restless manner, and frequently put the left hand to her head. Face flushed and inexpressive. Respirations 24. Pulse 60. November 6th, the breathing stertorous. Pupils variable, at one time contracted, at another dilated, without any external cause. Deglutition difficult; urine and fæces passed unconsciously. On the 8th she appeared more sensible, ate some bread and butter, and when spoken to made an attempt to answer, but was unable to articulate. The head was rolled from side to side, and the left hand lifted to it, as if in pain. On the 9th the catamenia appeared rather profusely, and she so far rallied as to recognise a relative who visited her, and to say distinctly, "My cousin." On the evening of the 10th her symptoms became aggravated, the face flushed, the eyes suffused, pupils dilated and fixed. Respirations 26, with an occasional prolonged expiratory effort. Pulse 72. Twitching of left side of face. Death a few minutes after this report.

Post-mortem examination.—On removing the membranes of the brain the anterior convolutions of both hemispheres were evidently flattened.

Under the arachnoid, on the left side, there was extravasation of blood, filling the sulci between the convolutions and blocking up the fissure of Sylvius. On making a section through the centrum ovale majus, the substance of the left hemisphere, external to the corpus striatum and thalamus opticus, was found softened to a great extent, and in the midst of this softened portion recent coagula and bloody serum. The effusion had not extended into the ventricles. The middle cerebral artery, on the left side, had upon it two small aneurisms. One of these had ruptured by a circular opening and given rise to the hæmorrhage. The descending cornu of the left lateral ventricle contained bloody serum, and the tissue was softened in many spots. Heart small, covered with fat; muscles soft and greasy. Some thickening of the mitral valve. Kidneys and other viscera healthy.



Remarks.—The length of time from the rupture of the sac to the fatal termination was explained by the state of parts after death, for the hæmorrhage, though extensive, had not broken in upon the central parts, but was spread out under the arachnoid. The remission of the symptoms on the fifth and sixth days was remarkable, and may have been due to changes in the clot, the pressure on the surrounding parts becoming equalised by its contraction and by absorption of the serum which had diffused itself through the softened tissue.

CASE 5.—*Ingravescent apoplexy; convulsions; atheroma of cerebral vessels; very large effusion of blood into right hemisphere; aneurism of middle cerebral artery on left side as it lay in the fissure of Sylvius.*

Mr. P—, æt. 58, of intemperate habits, and recently the subject of delirium tremens, whilst reading on Sunday evening, February 28th, 1859, dropped his book from the left hand, and his speech became indistinct. There was no exclamation. After an hour a violent convulsion came on, affecting the right side only. Convulsions returned four times at the interval of an hour or so, always limited to the right side, the left being slightly flexed and rigid. When the convulsions ceased he was able to answer questions, though slowly and indistinctly, and pointed to the right temple as the seat of pain. When visited five hours from the commencement of the seizure he was lying supine in a state of semi-coma, from which he could be partially roused; breathing stertorous; pupils contracted, only slightly acted on by light; optic axes divergent. Pulse 112. The following day, at one o'clock, he lay in the same state, and still pointed with his right hand to his forehead when asked if he had pain, and even muttered a few words indistinctly. The left side rather rigid and motionless. Priapism. In the evening the breathing became slower, and the coma more profound. He died forty-four hours from the beginning of the attack.

Post-mortem examination.—In the right hemisphere, external to the corpus striatum and thalamus opticus, and not breaking through either to the surface or into the ventricle, was a large effusion of blood, which had formed a cavity four inches in its antero-posterior extent, two inches and a half transversely, and one inch and three quarters vertically. The cerebral vessels were extensively atheromatous. In the fissure of Sylvius, on the left side, the middle cerebral artery was dilated into an aneurismal pouch the size of a large pea. The coats of the vessel forming the sac were transparent, nor was there any trace of lesion in the textures around. Its presence was a mere coincidence, and from all appearance it may have existed as an original deformity.

CASE 6.—*Headache at intervals for five years, sometimes severe; vertigo; tinnitus; sudden convulsive seizure, rapidly passing into coma; death in eight hours. Aneurism on anterior cerebral artery; rupture of sac; large effusion of blood over surface of brain and into the ventricles.*

Mrs. V—, æt. 35, of a delicate, rather spare, and anæmic habit, had for five years been troubled *at times* with headache, vertigo, indistinctness of sight, a sense of thickness in the ears, with tinnitus, and occasionally a noticeable slowness in apprehending what was said to her, though the intellect was clear. There was no such distinctness in her symptoms, however, as to lead to the suspicion of any organic disease in the cranium. Her ailments seemed to be referable to more general conditions, associated with slight anæmia and constipation. About eighteen months before her death she had an attack of headache, which continued for a fortnight, and prevented her leaving her room. The pain was never referred to any given spot, nor to the right or left temple; more commonly, in describing it, she put her hand to the occiput. Catamenia normal. My friend Mr. John Burton, of Blackheath, whose patient this lady was for a few months preceding the fatal seizure, informed me that her principal symptoms were vertigo, stuffing and ringing in the ears, a sense of general weakness, and constipation, and that she was apparently relieved by the use of iron and aloes, and by syringing the ears. The day before the fatal attack she was in her garden planting seeds, and was more cheerful than usual. At noon the next day she was taken with vomiting, and soon after fell into convulsions. At 2 p.m. the convulsions had ceased. She lay in profound coma, with the right arm flexed and rigid, and the right pupil dilated; the left side was flaccid and motionless, the left pupil contracted. Towards death both pupils became dilated and the trunk universally paralysed. Death at 9 p.m., eight hours from the beginning of the attack. It only subsequently transpired that this lady had had a fall from her horse a few weeks before her symptoms began.

Post-mortem examination.—The brain, both on the surface and in the ventricles, was inundated with blood, which had escaped from a rent in an aneurism of the anterior cerebral artery of the left side. The aneurism extended over to the right side, lying over the optic nerves. The sac at its distal part, on the right side, was formed by coagulated fibrin, and by the under surface of the inner angle of the anterior lobe of the cerebrum. The rupture had so occurred as to lacerate that portion of the brain which formed the floor of the third ventricle. The ventricles were full of blood, and the subarachnoid tissue infiltrated over the whole surface, but principally about the base and between the hemispheres. The brain-substance was nowhere destroyed but at the part indicated. The cerebral vessels were generally healthy. No spots of atheroma. The principal part of the sac of the aneurism was transparent, and formed of the normal coats of the vessel distended, but at its distal part, under the right hemisphere, the coats had given way, and the sac was formed, as described above, of a thin layer of fibrin and of the brain-tissue adjacent. The direction of the aneurismal dilatation was upwards and to the right side, in the course of the arterial current, and thus the optic nerves escaped pressure. The line of rupture also lay in the direction of the arterial current—namely, forwards and upwards, the effused blood tearing the floor of the third ventricle, and distending the meshes of the pia mater. The aneurism had been evidently for a long period of its existence what is called a true aneurism.

Remarks.—There was one symptom in this case which indicated more than others some organic disease about the brain—the occasional noticeable slowness in apprehending what was said. Slight as it seems to have been, it was in its character of the highest importance. In what way a local cause like aneurism should so affect the whole cerebrum is not to be explained until we know more of the nerve-force; but, as remarked above, we may suppose a local lesion capable of altering the polarity of the adjacent nerve-tissue and the condition of the rest.

DISEASES OF THE NERVOUS SYSTEM.

(*b*) DISEASES OF THE SPINAL CORD.

THE GULSTONIAN LECTURES.[1]

DELIVERED AT THE ROYAL COLLEGE OF PHYSICIANS, 1848.

LECTURE I.

ON THE NERVOUS SYSTEM.

Mr. President,—In appearing before the College to fulfil the duty which has devolved upon me by your appointment I cannot but express the sense I have of the honour conferred upon me, and my regret at the small means at my disposal for the due performance of the task. My previous engagements have left me but little leisure for new investigations. I am, therefore, afraid I can bring before you little that is new. I have chosen out some points in the physiology of the nervous system, particularly in reference to an arrangement of paralytic and anæsthetic affections. In this first lecture it will be my endeavour to set forth the present state of our knowledge in regard to physiology only; in the next lecture to consider some facts relating to paraplegia; and, in the third lecture, the phenomena and law of hemiplegia, with the exceptions. Of the importance of the nervous system we may form a just estimate when we remember, that in the development of the embryo of the higher animals, it is the first which appears. (The lecturer illustrated this by referring to a diagram of the embryo of the dog.) The first traces of the vertebrate form are in these simple lines of nervous substance; this is the centre in relation to which all the parts of the future animal are to be laid down. The nervous system has this priority, not only in fœtal development, but manifests it throughout the whole life of the individual. On reviewing the phenomena presented to us by the higher animals, we are led to the conclusion that their vertebrate organisation is an adaptation especially for action, being a co-ordinated system of nervous centres, nerves, muscles, and bones. The digestive system, corresponding to the mucous layer as the nervous does to the serous, is necessary for receiving the nutriment from without, and for elaborating it into the constituents of the blood, whilst the intermediate system of blood-vessels conveys the renovating and nutrient fluids to the first great system, and removes the worn-out tissues; but these two systems, the digestive and the vascular, are subordinate in the idea of the vertebrate type.

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[1] Reprinted from the 'Medical Times and Gazette,' 1848–9, vol. xix, p. 371, *et seq.*

In each segment of this great system we find several elements, each of which has its own function; although so great is the unity of the whole that no part can suffer without affecting the rest.

The muscle represents many units of power, according to the number of its fibres, many muscular fibres being submitted to the influence of one nerve-fibre, and many nervous trunks inserted into one common centre—*gradual concentration and subordination for the purposes of arrangement.*

The muscle has its own inherent and proper power of contraction in virtue of its organisation, and nerve is the proper exciter of this power, in a manner not explained; all we yet know being this, that a nerve, when mechanically disturbed, or affected with an infinitesimal amount of electricity, brings out the function of the muscle. We can compare the phenomena to nothing so aptly as to a spring set free by the easy motion of its stop. On irritating the nervous centre we no longer obtain the same simple results, but the phenomena give us the idea of SYSTEM, not merely *sum of units*, but UNITY OF PLAN.

The function of each part is obviously distinct; yet, in the higher animals especially, there is a most intimate combination and dependence, from muscle to nerve and from nerve to centre.

The advances of anatomy have demonstrated this in respect of the three elements. It is more than conjecture, at least in many instances, that the grey caudate cells of the nervous centres are continuous with the nerve-fibres, and the ganglionic masses on the posterior roots of the sensitive nerves in the lowest Vertebrata, where the tissues were so loose as to permit a successful inquiry, have been shown to arise from a vesicular dilatation of the nerve-tubules in their course.

Of the mode of termination of the nerves in the muscles and skin, facts of the greatest interest seem to have been fully ascertained. In respect of muscle the primitive nerve-tubulus, after reaching its surface, has been found to divide into two, or often many branches, instead of forming loops with fibres returning into the trunks from which they were sent off, as described by Breschef. There is yet, however, uncertainty as to the precise manner in which these branchings of the nerve-fibre end in the muscle ; yet is there much probability of *direct continuity.*

In respect of the skin and the cellular parenchyma of organs, there is at least a very close intermingling of nerve and fibre-tissue, if not absolute continuity of structure; so that it might not be too bold an expression, however illogical, to say that they formed an extended peripheral centre.

Passing onwards from these established truths, we come to consider the central parts of the nervous system; and the first question for our consideration is their symmetry. Is there one general centre, or are there many,—and if many, what is their arrangement and mutual relation ? The second question is,—Does the same law hold good in regard to centres as to nerves ; namely, is there one collection of grey matter for sensation and another for motion ?

In reference to the number of centres and their arrangement, the most striking theory is that proposed by Dr. Hall ; namely, that there is a true spinal system, made up of many segments, and a distinct sensory and volitional one.

That the spinal cord is more than a collection of nerves running to or from the brain has long been maintained. The movements of decapitated animals have led to many speculations; some of the older physiologists regarding them as the result of a residual irritability of the muscles, others thinking they arose from nervous sympathy; and Prochaska and Le Gallois, with others, regarded them as the result of a reaction on the spinal cord, in which the latter, at least, admitted sensation to exist. Dr. Hall has more fully than anyone else investigated these movements, and explains them, and a series of others in our own bodies, by referring them to a simple physical reaction, from which sensation is excluded as a necessary part, though often conjoined; whilst other movements, the results of volition and sensation, are carried on by quite a distinct system. However suitable such a supposition of two distinct sets of nerves might be to explain many phenomena of nervous action, yet are there many great objections to the hypothesis. In the first place, they must be equally extensively distributed in the skin and muscles. For as, in a decapitated animal, there is no part of the surface which cannot excite the spinal system, so is there no part, when the animal is entire, which, when injured, does not give rise to sensation. Again, the movements of the muscles are performed after the same manner, whether the muscles are acted upon by volition or by a physical reaction of the cord only. Hence we cannot but infer that the relation of the excitor and of the volitional fibres to the muscular tissues are the same. For instance, in a decapitated frog the legs are drawn up when pinched; so are they when the animal uses them under volition. The extension of these two systems, therefore, must be equal, and their arrangement similar; but to what purpose is not evident, inasmuch as we shall see afterwards that one set of fibres only, viz. the spinal set, would suffice. Another objection, which has seemed to me pressing against the theory of a volitional and an excito-motor set of fibres, arises from the power we have over these automatic movements. Take, for instance, the respiratory movements: we can arrest them at will; and in this we find a dilemma,—for, if we assume that the volition acts directly upon the muscles, the influence so sent to them *can be no other than a stimulus;* and if we cut off the stimulus of volition by a negation, we leave the muscles acted upon by the reflected irritation from the cord, which is powerful enough to excite them: hence they would continue to contract, and the volition would be powerless to stop them. But, if it be admitted, that the centre of volition can act directly upon the spinal centres, arresting the course of the impression from the incident to the motor nerves, then we can readily understand the manner in which we arrest the movements; but then the volitional fibres running from the brain to the muscles are *unnecessary*; for, if we can excite the muscles by acting directly upon the centres of the spinal cord with which they are connected, there can be no necessity for a double set.

Dr. Todd has well set forth, in his various writings, our ignorance of the exact arrangement of the nerves in the cord, the improbability of the longitudinal fibres being continuous with the nerves; and, after a careful repetition of the dissections of Mr. Grainger, arrives at the conclusion, that considerably the greatest number of fibres pass in at right angles (to the segments of the cord), whilst those which might be *supposed* to take an upward course are *few* and *indistinct*. The results of all this investigation being, that there is no satisfactory evidence of the cerebral fibres.

I have quoted the remarks of this excellent observer first, because his name is a great authority for their correctness; but will now add other evidence, which has been brought forward, and which tends to confirm this view.

As regards the tracing of fibres, a great source of fallacy arises from the softness of the tissue of the nervous centres, and from the lateral adhesion of the fibres themselves, so that with these difficulties before us, nothing less than an actual tracing of primitive fibres by the aid of the microscope could be certain evidence; and this has never been done to any appreciable extent. We must, therefore, form our conclusions as to the general arrangement of the nervous structures from more obvious conditions.

Volkman adduces, as a proof that all the nerves do not arise in the brain, the instance of the crotalus mutus, in which he numbered 221 pairs of spinal nerves. He proceeded to a careful measurement of their united area, and found that it surpassed by eleven times the area of the cord at its superior part. Sir C. Bell admitted that the respiratory nerves arose from the cord, and we have in the musculus accessorius what Bacon would have styled a *glaring instance* of spinal origin; for we can give no account of the strange course it must take, if it has a cerebral one.

If we select any animal remarkable for the muscular power of any segment of its skeleton, we shall find, as has been commonly stated, a corresponding development of the cord; and, as two such examples, I show you the cord of the jerboa and turtle. The same exists in the cetaceans which use their tail as the chief propelling organ, although some, speaking from theory rather than observation, have said that it is absent when the members are absent. As further tending to illustrate the arrangement of the nervous centres, we may refer to the present prevailing views of the symmetry of the vertebrate skeleton.

If it be proved that the skeleton is developed according to a definite plan of longitudinal repetition of simple elements, every segment or group of segments being modified in relation to the whole, whereof the proof is given in the harder parts of the animal structure, and the law and idea of the archetype traced out therein, on a due consideration of these facts, it must seem obvious that what holds of them will hold of the nervous centres, of which they are the mere supports. So that if we may recognise in a vertebra of the back a unit of the whole skeleton, limited or extended in the development of its parts, according to particular wants of the whole animal, we may, *a fortiori*, admit the spinal segment of the cord corresponding thereto, to *form a unit in the nervous mass*; and the same segmental or zonal symmetry which has, in modern times, been so successfully made evident in regard to the bony frame-work, must be admitted to obtain in the nervous centres, in relation and subordination to which all other parts are formed.

If we, therefore, reason from the known to the unknown, we shall be prepared to find in the nervous system the same kind of relations as in the vertebrate skeleton itself,—the same in kind, I say, but with a closer correlation than is even there apparent. There can be no more objection, *a priori*, to a mechanical arrangement of the nervous and muscular systems adapted for motion than to an optical apparatus for concentrating and absorbing the rays of light in seeing. This would, in fact, constitute the true spinal system of Hall; and, as Dr. Todd remarks, we must regard the decussating fibres of the anterior pyramids as commissural only.

We have, in the spinal cord, with its nerves and their conjoined muscles, a mechanism which, under ordinary conditions, is submitted to our volition, as in the segments corresponding to the arm and leg, or which can act automatically and yet be subject to volition, as the segments ministering to respiration, or which are mechanical only, as deglutition. And when we consider in detail the degree and kind of power we have over our muscular system, these views seem to me greatly confirmed. As for example, I may revert to the volitional power over respiration. How do we produce this complex movement? Not by our volition having any power to select out one or two individual muscles, nor even the muscles of one side,—but we produce the result *as a whole.* Nor is there any necessity for all these fibres to run to the brain, seeing that when they arrive there, as our common experience tell us, we have no power of selection over them, and particularly as one excitor fibre, or other equally simple means, from the brain would suffice, as proved by the simple fact, that such an irritated fibre does suffice to set the whole respiratory system into consentaneous action, as in sneezing, which may be set up by an impression conveyed through one fibre. But if this example be objected to we may instance those parts of the body which are most obviously under the influence of the will, say the arm or leg; the same inability of directing our volition upon any one muscle obtains also here. We will a particular movement, for example, flexion or extension of the arm, or adduction of the thigh, and we get the result without any obvious combination of elements. How numerous soever the muscular fibres of any one muscle, or of the muscles themselves, for a given result, we use them in sets. Nor can I see any obvious design in all the nervous fibres from the flexors or extensors, or from any one muscle going to the brain, seeing that one or two fibres, or any equally simple communication between the brain and the segment or segments of the cord with which they are connected would suffice.

All considerations of the mode in which we use the muscles, and the power we have over them, would lead to the conclusion that the nerves are arranged into centres, and that we act upon these by our volition rather than upon the muscles, individually or directly. I believe this view of the subject will also be further strengthened if we consider the anatomy of a plexus and its probable uses.

It has commonly been said that a plexus of nerves consists in an interchange of fibres of sensation and motion, for the purpose of transmitting the sensitive to the skin, and the motor fibres to the muscles; and it is a curious anatomical fact that the nerve which supplies the muscle supplies, for the most part, the skin over the muscle, according to Mr. Hilton's dissections. Dr. Todd hints at the general use of a plexus for motion, but by no means develops his idea respecting it. Reverting again to the kind of power we have over the muscles, and the fact that plexuses are complicated in relation to the variety of movements of the parts supplied by the nerves subsequently sent off from them, it would seem that the use of a plexus is to bring the different segments of the cord into relation *with groups of muscles*. Thus, let A, B, C, D represent so many segments of the cord, A being in connection with one group of muscles, B with another, and the same with C and D; by acting upon the centre A we get a result corresponding to its connections,—we can conjoin it with B, or C, or D, or with any one or all of these, and get so many and so varied movements, as, for example, flexion with supination, equable throughout the entire act of flexion, or supination first and then flexion, or flexion first and then supination, and so with every other possible muscular movement, by conjoining the various segments, and acting upon them in different times, as we should play on the keys of an instrument.

A plexus, therefore, must be regarded as a necessary part of the segmental symmetry of the cord—a necessary condition for combination and arrangement, although, on the hypothesis of our acting directly upon the muscles, one can see no necessity for such an intercommunication of the roots.

As confirming this view of the use of a plexus, I may refer to the paralysing effects of injury of one of the roots of a plexus. If a nerve be divided before entering a plexus we do not produce paralysis of any one muscle, but a general diminution of power in the whole.

As still accumulative evidence of this zonal symmetry of the nervous system, I would submit to your consideration the facts so notorious of the crossed action of the hemispheres on the cord.

It is well known that injury of one hemisphere paralyses the opposite side of the body; but there is at least one exception, and that is the third nerve, which is under the influence of the hemisphere of the same side. I shall give evidence of this in my remarks on hemiplegia. On considering this curious disposition of parts, the first question that forces itself upon the mind is, why is there a decussation at all? and, secondly, why is the third nerve an exception?

Proceeding upon the belief that the spinal system is chiefly for motion, consisting of many segments, whose parts have a definite arrangement and relation, we are naturally led to inquire into the means whereby we are enabled to employ it. It will be readily conceded, that a previous knowledge of the mechanism itself can be no guide to us, inasmuch as but few persons have any, even the most vague, idea of its existence; and with those who have an anatomical acquaintance with it, a contemplation or consideration of its parts forms no link in the chain. The phenomena of volition are *intention—result*, the sensation guiding to the end without any calculation of the aptness or adjustment of the means.

The will produces its effects under the guidance of sensation. I would illustrate this by referring to the muscular movement of the eyes and the phenomena of vision. The eye is directed to objects by two pair of nerves, the third and the sixth, the former being, as I have stated above, an exception to the crossed influence of the brain, and, as it would seem, for a very mechanical reason. Suppose any impression affects the two eyes equally, the third nerve and the sixth adjust the eye to a due convergence upon it, and we get an equable motion of the two globes; but again, let the object be placed on the right or left side: we now require, in order to direct the eyes to it, the conjoined action of the third nerve of one side with the sixth of the opposite. As the optic nerves manifest a decussating result, the object being most distinctly perceived by the nearest eye, for illustration, say the right, will transmit its impression to the left hemisphere, which will re-act upon the third nerve of the left side and the spinal centres of the right, comprising the sixth; both eyes will be immediately directed to the right, the head turned thitherward also; and, if necessary, by a movement of the right arm any approaching injury repelled. But if we suppose any other disposition, viz. that there should be no decussation, or that the third nerve should not be an exception, the beauty of the existing arrangement becomes especially obvious. As the body is now constituted if the object affects both eyes equally, and, consequently, both hemispheres equally, we have a conjoined action of the recti muscles of both eyes; if, by the lateral position of one object, one hemisphere be affected, which will, from the decussation of the optic nerves, be that opposite to the object, we must have such an arrangement, that this hemisphere can act reversely on segments of the cord. Hence do we find all the phenomena placed in a beautiful circle of relations. Thus, proceeding from the external object, the ray of light affects the nearest eye; this, transmits its affection to the opposite hemisphere; this, in its turn, reacts upon the spinal segments in connection with it, viz. the third of its own side, and the segments of the opposite turning both eyes, and directing the upper extremity, if necessary, to the object, and returning to the point whence we set out.

In regard to this decussation, there is another fact worthy of consideration and which has been spoken of by others, viz. that the nerves placed above the point at which the decussating fibres cross from side to side, are as much subject to the law of inverse action as the nerves below. The seventh is an example. That this should be so, cannot be adduced as anything strange, if we regard the symmetry of the segments after the manner here maintained; for as such a crossed influence seems even necessary, as I have striven just now to show, it remains to consider what part would be most appropriate for the placing of the commissural fibres; and out of this arises a principle of great interest in regard to the *exact point of the decussation.* It is well known that hemispheric lesions, producing hemiplegia, affect the upper than the lower extremity, and the seventh and eighth nerves with the lingual are generally less affected than the nerves of either the arm or leg. The paralysing influence is, therefore, not equally distributed through the opposite side; it is felt most severely in the parts which lie in the direction of the decussating fibres. Hence, had the commissure been from the summit of the segments of the spinal system and the same ill effects followed as we now know to result from cerebral lesion, would there not have been a much greater depression of the respiratory centres under cerebral injury, than is now produced? And is there not a conservative adjustment in this particular position of the decussation, as well as in the decussation itself by which the respiratory centres are *placed out of the line of action of the hemispheres, on the segments of the cord.*

The relation of sensation to the reflex movements is in the higher animals a complicated one; and, whilst we admit the principle laid down by Hall, that it does not form *any necessary* part of excito-motor phenomena in their simplest condition, yet are there others in which the sensation is intimately and essentially precedent to the movement.

The unity of organisation in man and the higher animals is such, that the condition of one part may react through the whole; and this is strikingly demonstrated in the nervous system, in the central parts of which, although the same symmetry and anatomical relations may exist as in the lower animals, yet there is not by any means the same physiological independence. In the development of the parts of the human body, there are many relations shadowed forth which tend to show that it has a symmetry like the lowest animal forms; and there is coincident with these immature states, the same independence of function. I call attention here to these well-known facts as confirmatory of a distinct spinal system; for, whilst we must admit, that in the adult the nervous segments have less independence than in the fœtus, as proved by the phenomena of anencephalic monsters, and the results of craniotomy; yet we may not deny to the adult the same symmetry as the fœtus—the same arrangement of the active parts. I might, perhaps, adduce the fœtal movements in proof of the excitability of the spinal centres, for, I think, they cannot be regarded as voluntary. If it be true, that the tissues in their development pass through phases of lower organic forms, we may expect the same independence of action to be coincident with these transitory conditions, as is found in those permanent states. I had a striking confirmation of this principle in the heart of a fœtus of the fourth month. It was expelled prematurely about 5 o'clock one summer's afternoon, and, at 8 o'clock in the evening, I proceeded to open the thorax, in order to inject the minute terminations of the bronchial tubes, and, to my surprise, I found the heart to possess some irritability; it contracted on being pricked with a needle after the manner of a fish's or reptile's heart when it has become so exhausted as to have lost its rythmic movements. This muscular irritability was like that of the cold-blooded animals; nor can we doubt that its spinal segments had the same relations.

There is a close subjection of motion to sensation in the action of the muscles of expression. I refer to those movements which have been called emotional, and which are as directly *excito-motor* as any, with this peculiarity, that emotion, or sensation, forms a necessary part. If we style the spinal movements *mechanical*, they are *psychico-mechanical*.

The combination of sensation with voluntary muscular movements has, by Volkman, been shown to be the source of our knowledge of locality and direction. It is not to my purpose here to consider how he has applied this to visual direction and position of objects; but I may just mention, that it has long seemed to me obvious, and I have long so taught it in lectures, that, as it is by the muscular movements of the upper extremity that we test the direction of any force acting upon the sensitive nerves of the fingers, so it is, by the contraction of the muscles attached to the eye, that we tell the position of any object which sends its rays to the retina. If the object be placed above, we use the superior recti; if below, the layer; if to the right, the muscles of the right side.

The great discovery made by Sir G. Bell, that the posterior nerves of the cord were for sensation, and the anterior for motion, led him to express his belief that the columns of the cord itself had corresponding functions. Nor would it be easy to admit the contrary, if there were any grounds for believing that the columns were continuous with the nerves, and formed by them; but as I have, at the beginning of this lecture, attempted to prove such an opinion is not founded upon observation, and although experiments devised for the proof of this opinion may seem to be affirmative of the separate function of the columns, yet one must object to them as extremely fallacious, considering the oblique origin of the nerves from the segments of the cord. Nor are experiments at all conclusive, inasmuch as very different results have been obtained. Amongst modern physiologists, such an exclusive function is no longer maintained; but as yet no general law has been enunciated which shall apply to pathological conditions. I am of opinion that the cases on record of local lesion of the cord will warrant the conclusion that the law demonstrated of the nerves does not obtain in the columns; that neither the anterior nor the posterior columns have special functions in reference to sensation or motion. And whilst, on the supposition of continuity of fibres upwards in the cord to the brain, such a separate office of the columns seems inevitable, so, on the other hand, if it can be proved that they have no such function, we have an argument for the segmental symmetry and for that theory which is now generally admitted, of the fibres of the cord being commissural, uniting and weaving into a system these many centres for muscular action, and adapting them to our use.

From this general survey, I hope, in my next lecture, to pass to the consideration of paraplegia; and my endeavour will be to show, that there are at least three forms of this affection, which are accompanied with distinctive symptoms, and that the phenomena presented by paraplegia greatly favour this zonal and independent symmetry of the spinal system and the commissural function of the columns.

LECTURE II.

PARAPLEGIA.

One of the chief objects of this inquiry into the phenomena of paraplegia which I propose to myself, is to show that there is, according to its different forms, a different degree and kind of anæsthesis; in some cases the loss of motion being nearly complete, with but little and always less affection of the nerves of common sensation; in others, the loss of sensation preponderating over that of motion.

That in paraplegia, resulting from local lesion of the cord, *whether situated in the anterior or posterior columns, or affecting the whole substance equally*, there is more paralysis than anæsthesia. That when there is preponderating affection of the nerves of sensation, we may not anticipate that a post-mortem examination will show any limited lesion of the cord in any segment; for in such cases the disease often arises from peripheral changes in the nerves, or from conditions of the central organs not recognisable by our usual methods of investigation, yet often associated with morbid accumulations of fluid in the ventricles of the brain, subarachnoid space, and opalescence of the membranes.

Paraplegia, arising from injury to the cord through mechanical violence is extremely common. For the most part, the effects of the injury are immediate, motion and sensation being for the time, greatly affected or entirely lost. If the lesion be less than to produce division or disorganisation of the whole cord, although there may be considerable anæsthesia immediately following the accident yet the sensitive nerves, after a time, recover their function, though the paralysis may continue; and this mode of recovery is, in such cases, universal.

Before giving the records of the simplest cases of paraplegia which prove the positions I have laid down, I shall quote the experience of Mr. Earle, whose excellent article on paraplegia (in the thirteenth volume of the 'Med.-Chir. Trans.') I shall again have occasion to refer to. "In spinal affections," says he, "sensation will often remain perfect after a total loss of the locomotive powers." So Sir B. Brodie, "when recovery takes place the restoration of insensibility usually precedes that of the power of voluntary motion, so that the patient may be quite sensible of external impressions while he is still incapable of employing his muscles for any useful purpose. The last observations apply equally to all cases, whether the spinal cord has suffered from concussion or from the pressure of displaced vertebræ."

One of the great causes of our little advance in the study of nervous affections seems to me to have arisen from a tacit assumption that the phenomena were too uncertain and varying to admit of any general expression. Dr. Abercrombie seems to have striven to place, side by side, cases which should set at defiance all law, nor has he attempted to reconcile the discrepancies. But all will admit that to rest satisfied with this would be to take too narrow a view of the subject, for here, as elsewhere, there can be nothing variable; the order and relation of the phenomena must be as definite as in the inorganic world, though the conditions may often be so complex and interwoven as to elude our explanation. What is true of one case must be equally true of another like it; yet this truism has hardly been admitted in nervous pathology. My endeavour, therefore, to establish some general laws, if fruitless, must still, I think, be in the right direction.

In paraplegia arising from the lesion of the cord, whether the anterior or posterior columns, or the cord generally, be affected, there is greater loss of motion than sensation. In proof of this, I shall refer to several well-known cases of disease of the columns. I may be allowed to premise, that it did not seem to me necessary to give cases in the full details with which they have been recorded by their various authors, for other purposes, although I have been careful not to omit any particulars which could bear upon the question here in consideration. In the 'Medico-Chirurgical Transactions,' for 1842, Dr. Webster gives a case of paraplegia, arising from inflammatory softening of the cervical portion of the medulla spinalis, affecting generally the whole structure of the cord and its membranes; although from minute investigation of the diseased parts subsequently by Dr. Todd, it seemed that the posterior columns had most suffered. The individual affected had been, for many months *totally deprived of the power of motion* below the seat of the disease, *yet his sensation was perfect throughout the entire surface of the body,* with the exception of an over-sensibility of the affected parts, towards the end of the malady, which, together with many subjective sensations of heat and cold through the surface, although there was no change of the temperature of the external medium, excited the curiosity of those who watched the case, expecting to find some particular portion of the cord alone affected. As the writer tells us in his remarks on the case, "This instructive and interesting example of disease of the nervous system excited, as well it might, much attention in all those who witnessed its progress; and they, along with myself *confidently hoped* that the pathological appearances met with on dissection would be such as to explain satisfactorily the rationale of the peculiar symptoms which the malady exhibited during the patient's protracted and severe sufferings, more especially in regard to those remarkable features characterising the case, namely, that whilst sensation remained unimpaired, voluntary motion was totally suspended; because, should these anticipations be realised by the post-mortem examination, some important physiological truths would have been elicited. It is clear from these observations that an impression existed in the minds of those who witnessed this case, that the columns did subserve to motion and sensation; but the result of the post-mortem examination by no means favoured such a theory, for not only were the posterior columns greatly diseased, but there was a general affection of the whole cord and its membranes.

James P—, æt. 58, a farm-bailiff, was admitted into Guy's, under Dr. Babington, in the year 1845. He had enjoyed very good health until the autumn of the year previous, it now being spring; he then began to complain of weakness in the legs and spasmodic constriction of the abdomen, as if a cord was passed tightly round the waist; he never had a fall, nor any other injury to the back. On his admission there was not entire inability to move the legs, but they were very weak, the degree of the paralytic affection varying. One of the points of most obvious remark was the tendency to spasmodic action of the diaphragm under emotions, or on touching the abdominal walls. *There was no perceptible diminution of sensation.* He was treated by nervine tonics, as zinc, together with counter-irritation by the ferrum candens, and the progress of the case was marked by variable improvement and relapse, the power over the legs becoming entirely lost, but the sensibility not being to any marked degree affected; and, on a post-mortem examination, there was no lesion of the brain nor of the parts about the spine. The entire substance of the cord in the middle of the dorsal region was soft and diffluent, and of a dull, opaque-white colour.

The case recorded by Mr. Stanley, and which has been so often quoted, may be adduced here in proof of the assertion, that disease of the substance of the cord uncomplicated, produces greater loss of motion than sensation.

The patient was a man aged 44. The disease had begun gradually without any previous injury, and resulted in complete inability of motion, whilst there was, at the same time, not the least discoverable impairment of sensation. The substance of the cord through its posterior half and columns, and in its entire length from the pons to the lower end, had become of a dark brown colour, and was very soft, whilst the anterior part had its due colour and consistence. The membranes were healthy, with a slight excess of the rachidian fluid. Mr. Stanley remarks:—"We cannot, in the present state of our knowledge, satisfactorily transfer the same view of the distinctness of function of the corresponding columns of nervous matter composing the spinal cord (as obtains for the roots of the nerves)." It is the object of these remarks to show that such a law does not hold."

Dr. Budd has recorded the following case:

R. H.—, sailor, aged 17. In August, 1837, received a severe blow on his back from the boom of his ship, which did not, however, disable him; he continued his work as usual. From that time he suffered occasional pain of the loins, and weakness in the back, especially when stooping. This continued, without other complaint, until the beginning of December, when he began to experience difficulty in running.

On the 8th of January he was admitted into the Seamen's Hospital. On admission his lower extremities were observed to be in extension and very rigid, with sensation unimpaired, except slight numbness of the thighs.

In the beginning of April profuse hæmoptysis took place, and was soon followed by other symptoms of phthisis. His intellect had continued unimpaired; the lower extremities quite deprived of voluntary power, but *with sensation unaffected.*

On examination after death, the curvature of the back was found to be formed by prominence of the dorsal vertebræ, from the fifth to the ninth inclusive.

As soon as the laminæ of the vertebræ were divided, a small quantity of yellow pus oozed out from the right side of the spinal marrow, exterior to the dura mater.

The dura mater, for the space of an inch and a-half corresponding to the most projecting portion of the spine, had a dark greyish stain externally, and was much more vascular than the portion above or below the curvature.

The diameter of the cord was considerably smaller in the portion corresponding to the curvature than in any other part throughout its whole length.

There was no pus in the sheath.

The cord was of natural size, and a portion about two inches in length, corresponding to the curvature, softened in the posterior columns. The tissue was not diffluent, but became flaky and partially dissolved when a small and gentle current of water was poured on it. This did not happen when a like current was similarly directed on other portions of the cord. This breaking of the tissue was *much more marked in the posterior* than in the anterior columns, which were scarcely, if at all, softened, and resisted considerable traction.

Under the heading "Undefined Suppuration of the Cord," Abercrombie gives three cases, in neither of which is there any mention of the state of the sensation, although, in his other reports of similar affections, he is careful to note when the anæsthesia has been to any degree marked.

I might go through every case recorded by this author, and show, that there is not one in which, from lesion of the substance of the cord, sensation was alone affected, or in which the anæsthesia prevailed over the paralysis. And here I would remark, that whilst the cases I have given have been detailed, in some instances, to show that the posterior columns were not for sensation, no general deduction has, so far as as I am acquainted, been drawn from them.

The following cases illustrate the same principle with variable seat of the lesion.

A man aged 36. Paraplegia from fall; complete loss of motion in lower limbs, without loss of feeling. *Post-mortem.*—Extensive ramollissement of body of cord, affecting anterior columns chiefly; the posterior columns softened in many places, though in smaller degree. (Abercrombie, 129th case.)

In a case by Olliver. Gradual palsy of lower extremities; patient bedridden for seven years; legs drawn up to the body, entirely motionless, but preserved their feeling. *Post-mortem.*—Extensive ramollissement of upper part of cord, especially in the anterior pillars and corpora pyramidalia.

In a case by M. Rullier. Gentleman, æt. 44. Curvature of spine, paralytic flexion, and at length complete palsy of arms, which became rigid, contracted, and entirely motionless; sensibility of parts not affected. *Post-mortem.*—Six inches of cervical portion of cord entirely diffluent, so that before membranes were opened it moved upwards and downwards like a fluid. This case is given by Abercrombie.

Man æt. 20. Complete paraplegia; no diminution of sensibility. *Post-mortem.*—Disease of third, fourth, and fifth cervical vertebræ; tuberculous disease of membranes at this part; ramollissement, chiefly of anterior columns; posterior slightly softened. Abercrombie, sect. v, case 3.

Gentleman æt. 18. Perfect paraplegia, without loss of sensation. "There had never been the least attempt at motion of the lower extremities, but the sensibility remained." There is mention of numbness in this case; but the quotation is in Abercrombie's own words, and this affection of the sensation will be subsequently considered.

It might not appear necessary to quote cases of affection of the anterior columns, as they have been regarded as normal when the power of motion has been most affected: but they equally bear upon the question I am considering with those in which the posterior columns were the seat of the disease; forasmuch as both being accompanied by the same symptoms, neither can be regarded as proof of the special functions of the parts. And whilst it must be admitted that paralysis existed beyond anæsthesia, when the anterior columns were affected, it cannot be inferred that this was because the anterior columns were for motion, inasmuch as the same occurred when the posterior columns were the seat of the disease.

The following case from Dr. Bright's reports may, I think, be adduced as bearing on this question, although he refers the paraplegia without anæsthesia to the position of the lesion; but the preceding remarks appear to me to show that such is not the explanation.

William B—, æt. 20, was admitted under Dr. Bright into Guy's, August 18th, 1844. About six weeks before, without any cause of which he was aware, he began to feel pain in his neck, close to the head, which had increased, with some stiffness of the part. At the time of his admission his head was bent forward, so that the chin approached the upper part of the sternum; there was almost complete hemiplegia on the *right side, and some* flying pain, which he called rheumatic. He complained of pain when the vertebræ of the neck were pressed, and there was an apparent displacement or slight irregularity in the spinous processes of that part. Leeches were applied, and local applications made to the nape of the neck.

At the end of November (three months) the other (the left) leg became much affected.

In January (five months) he was unable to walk without assistance. He complained that his legs *felt asleep, but they retained their sensation.* He died on the 8th of February, the report stating that on the day previous though quite sensible, he was unable to move any part of his body.

The principal post-mortem appearances were confined to the processus dentatus, which, by its enlargement and displacement, had contracted the foramen very much. At a point where the pressure was made, the medullary matter of the spine had assumed a darker colour, to the extent of about a pea, and was apparently vascular. It is to be remarked, says Dr. Bright, that whilst motion was destroyed sensation remained perfect—a fact which may be easily accounted for when we consider that the processus dentatus was the part chiefly diseased, and necessarily made pressure on the anterior part of the cord.

But, inasmuch as sensation remains perfect when the disease is general, or on the posterior part, this case cannot be adduced in favour of the columns having especial functions.

In the ninth Volume of the 'Med.-Chir. Trans.,' Dr. Bostock has given a case of universal paralysis, of which the following is an outline; and I quote it for the purpose of showing that general affection of the cord in this, as in the other cases quoted, manifests the same law in regard to anæsthesia and paralysis.

M. H—, a middle-sized and well-formed man, between 30 and 40 years of age. Had enjoyed good health. Applied for medical advice in consequence of a pain which he experienced in one of the lower extremities. Its seat was on the outside, and a little above the knee, but it sometimes shot up to the hip. He mentioned that he had had a slight fall, but it seemed so unimportant that he attached little to it.

After two months the power of moving the limb was perceptibly diminished, and in the next two the complaints continued slowly to increase. The limb possessed its full share of sensibility and was of the natural temperature, but he lost more and more the command over its motions. There was no pain in the trunk, nor could disease be detected in the spine. In two months more the other limb was similarly affected. There was no numbness, but shortly after this period a new affection made its appearance, —a difficulty of the articulation of particular words, and for the next four months the affection of the speech and of the limbs continued to increase, so that he became unable to move without assistance, and his power of utterance was nearly lost; and eight months afterwards he first complained of pain in the back of the head and neck, and the paralysis of the upper extremities and affection of the muscles of deglutition supervened. Notwithstanding this loss of voluntary power, there was *no numbness nor insen-*

sibility of any part of the body, neither to mechanical impressions nor changes of temperature. All the external senses and mental faculties remained unimpaired. He dragged on a wretched existence for six months, and died exhausted,—his senses, special and general, remaining unimpaired.

Post-mortem.—We began by examining the brain, and every part of it was most minutely scrutinized, but without our meeting with any appearance which could be considered morbid. After a very accurate survey of every part, we thought we observed a slight furrow *across the spinal cord, as if it had* been compressed by a transverse ligature, and this in a place where it passes under the ring of the atlas; and, upon attentively noticing this part of the bone, it appeared a little thickened and of a yellowish colour.

Very numerous cases are on record of pressure on the cord, from tumours, hydatids, &c., in all of which loss of motion accompanied loss of sensation, when this latter formed part of the symptoms; but I cannot find any cases of pressure or disorganisation uncomplicated, in which the loss of sensation preponderated, or in which there was simple loss of sensation.

My attention has now for years been much given to post-mortem appearances; but I have not met with cases which controvert my position, nor are there amongst the numerous cases of lesion of the cord which are contained in the records of this hospital, any which show that one part or column of the cord when affected, is attended with anæsthesia without loss of motion, though as we have seen, the reverse often happens.

If it is admitted that these facts warrant the conclusion that seems to me fairly to flow from them, we have therein another argument in favour of the zonal symmetry I advocated in my last lecture. If we admit that this spinal system, in its various segments, can act under the form of an excito-motor system, which necessitates continuity of action from incident to reflecto-motor nerve, we cannot, I think, even suppose in these segments two centres; one, namely, for receiving the impression from without, and the other for exciting the movement in the muscles. Nor does there seem to me any stronger grounds for admitting separate centres for motion and sensation in the brain than in the cord. How fallacious soever these remarks may be, yet the facts seem to oppose the assumption that there are such sensory and motor centres, distinct from each other.

In pursuance of my subject I have next to call attention to those cases of paraplegia in which there is much greater loss of sensation than of motion; and far from finding them contradictory of the former, or anomalous, they seem naturally to arrange themselves into distinct classes. Before treating of the two great divisions of such cases I would observe that in paraplegia from disease about the spine the nerves, as they pass to the cord, are sometimes implicated, and we find on a post-mortem examination sufficient explanation of what, carelessly observed, might seem to be an exception to the rule laid down. Thus in the case of the Count de Lordat, mentioned by Abercrombie, a paralytic affection of the left arm followed an injury to the neck from a fall, and the opposite arm became numb. On a post-mortem examination, together with induration of the cord, there was a *compact and tendinous condition of the cervical nerves, owing to thickening of the membranes covering them.*

In some cases of disorganisation of the spinal centres numbness or other affection of the sensation may precede or accompany the disorganising process and yet, even as the paralysis increases, the state of the sensation shall improve and become quite normal. It would seem, from the similarity of such cases to the results of concussion of the spine, in which the sensibility returns after a few days, as if the onset of the local disorder produced the condition of concussion, destroying for a time the power of feeling which, so soon as the first shock of the inflammation is over, returns again. This is strikingly exemplified in those gradually extending affections of the cord in which anæsthesia is the precursor of paralysis, and then disappears.

The first class of paraplegic affections, with preponderating anæsthesia, are peripheral, beginning as impressions on the extremities of the sensitive nerves, the muscular movements suffering subsequently. The efforts of modern physiology have been directed to the elucidating and setting forth the functional value of each particular part of our bodies, and its success has nowhere been greater than in regard to the nervous system. We no longer regard nerve as a mere conductor of an influence received from the spinal cord; it is in itself as active, according to its particular function, as the centre to which it is attached or the muscle with which it is connected. The prevailing views in physics will find their way into every branch of natural study; hence we talked for a time of the nervous fluid, and there were theories to show that the nervous fluid is probably a highly subtil one, allied to the electrical. But electrical fluids expressed no more than an hypothesis, and we now regard the conducting wire as so conducting by virtue of its active molecular forces. We alter its physical state and we alter its power of conduction, and this reacts through the series. This great advance in physics has gone hand in hand with the discoveries in neurophysiology. Nerve has the power of exciting muscle by virtue of its proper state of nutrition. This is proved by exhausting a nerve. After leaving it at rest for a time it recovers itself, although previously divided from its centre.

The nerves, in their distribution through the tissues, make up a very widely extended area of nervous substance, whose due nutrition and otherwise healthy condition is necessary to a proper exercise of its functions; and, if we also take into consideration the position of its extreme parts far removed from the centre of circulation, and its susceptibility, we shall be disposed readily to admit that, from its extent, its position, and its delicacy, that it deserves our closest attention.

Dr. Graves, who has treated on this subject, thus expresses himself:—"May not," says he, "the decay and withering of the nervous tree commence occasionally in its extreme branches? and may not a blighting influence affect the latter, whilst the main trunk remains sound and unharmed? Pathologists have, with respect to diseases of the nervous system, committed an error precisely similar to that which was so long prevalent with regard to diseases of the vascular system, for it is only lately that, in estimating the forces which influence the circulation in diseased parts, they have begun to appreciate the preponderating influence of the capillary vessels, independently of the heart's action and the *vis a tergo*. It is only lately that they have recognised the important truth, that diseased vascular action may commence in the circumference. He quotes as one of the most remarkable of such peripheral affections, the *Epidémie de Paris* of the spring of 1828, as follows:—"Chomel has described this epidemic in the ninth number of the 'Journal Hebdomadaire,' and having witnessed it myself in the months of July and August for the same year, I can bear testimony to the accuracy of the description. It began (frequently in persons of good constitution) with sensations of pricking and severe pain in the integuments of the hands and feet, accompanied by so acute a degree of sensibility, that the patients could not bear those parts to be touched by the bedclothes. After some time, a few days or even a few hours, a *diminution or even abolition of sensation took place* in the affected members; they became incapable of distinguishing the shape, texture, or temperature of bodies, the power of motion declined, and, finally, they were observed to become altogether paralytic. The injury was not confined to the hands and feet alone, but, advancing with progressive pace, extended over the whole of both extremities. Persons lay in bed powerless and helpless, and continued in this state for weeks and even months. Every remedy which the ingenuity of the French practitioners could suggest was tried and proved ineffectual. In some the stomach and bowels were deranged, and this affection terminated in a bad state of health, and even in death. In another, the vital organs, cerebral, respiratory, and digestive, were in the same state as before their illness, and their appetites were good, but still they remained paralytic. At last, at some period of the disease, *motion* and *sensation* gradually returned, and a recovery generally took place, although, in some instances, the paralysis was very capricious, vanishing and again reappearing. *The French pathologists searched anxiously in the nervous centres for the cause of this strange disorder, but could find none.* There was no evident lesion (functional or organic) discoverable in the brain, cerebellum, or spinal marrow.

The learned author from whom the above quotations are made uses these facts, with others, to prove that paraplegia may arise from diseased conditions of the nerves in their peripheral distribution; and, whilst they are here introduced for a similar purpose, yet the chief object is to show that, in such cases, the nerves of sensation are often affected to a greater degree than those of motion, contrary to what occurs in diseases of the cord. The following are illustrations of peripheral paraplegia from cold, with prevailing loss of sensation :—

David F—, æt. 50, was admitted into Guy's, under Dr. Bright, in March, 1838. By trade an engineer and wheelwright; muscular. His present illness began seventeen years ago, with an aggravation five months since. His first symptom was *numbness in the lower extremities*, extending as high as the hip-joints. He had been at this period much exposed to wet and cold; *the power of motion was not in the least affected*, and he continued to do his work without intermission. He could walk a distance of six miles without inconvenience. Five months since the numbness extended, and he attributes it to his having about this period been engaged in working in a damp well; after this, with the extension of the numbness, the power of motion in the legs became impaired, and the arms in some degree participated. After being in the hospital for a short time he had twitchings in the lower limbs, and the power over the sphincters was diminished. His symptoms increased to entire helplessness; his legs became œdematous, the urine highly ammoniacal, and he died worn out. There was no section of the body.

Although this case is incomplete, yet I think we may trace the peripheral affection of the nerves of common sensation from cold and damp lasting without muscular weakness for seventeen years, and under fresh exposure the nervous centres beginning to suffer, followed by paralysis and all the evils attendant upon ramollissement of the cord. I need not here insist upon the fact that the nerves do suffer from cold, and that at parts far removed from its direct application, as in sciatica from standing on cold stones.

The following case is given by Dr. Graves :—

James M—, was admitted into the hospital labouring under paraplegia, which he attributed to cold and wet. About a month before admission he first perceived a stiffness of the great toe of the right foot, afterwards *numbness* and coldness of the sole, and then of the leg as far as the knee, and dragging of the limb in walking. During the progression of the disease up along the thigh it commenced in the left foot, and after a few days he experienced *almost complete paralysis of sensation* in the right lower extremity, and a lesser degree in the left, accompanied by so much diminution of the power of motion as to render him unable to walk without support. About three weeks after the appearance of paralysis in the lower extremities the little finger of the right hand was attacked with *numbness*, which passed successively to the rest, attended by some loss of the sense of touch and power of grasping objects. He has also had retention of urine, and the bowels were obstinately constipated. There was no tenderness of any part of the spine; he had no pain in the head; his pupils were natural; mind unaffected; pulse, sleep, and appetite also, natural. "This case," says Dr. Graves, "I am convinced, had its origin in the extremities."

Having thus shown that cases of paraplegia, with prevailing numbness, may arise from causes acting on the nerves themselves, I shall next call attention to a third class of paraplegic diseases, resulting from anxiety, mental depression, irregular practices, and all attended with general diminution of the nervous force.

The relation of the parts of the encephalon to the segments of the cord, is one of great difficulty to determine. I have observed that emotions of a depressing kind generally affect the lumbar portion of the cord very greatly. I have now a friend who has suffered from extreme susceptibility of the whole nervous system, resulting from great intellectual exertion; and he describes to me that, under the least emotion, the shock felt in the loins is most appalling, seeming to take all the strength out of him. The trembling of the knees in fear is of the same kind. In the year 1820, Dr. Baillie published, in the 'Transactions of the College of Physicians,' "Some Observations on Paraplegia of Adults," in which he states that where the spine has not suffered from outward violence, paraplegia most commonly depends upon a disease of the brain itself. Now, although I think our present state of nerve physiology, and the facts I have just read, will not warrant us in arriving at so general a conclusion, yet certainly a fair number of cases of paraplegia have an encephalic origin.

Mr. Earle, in his excellent paper on paraplegia, made some most practical remarks on this form of the disease, and is the only writer, so far as I know, who has pointed out the condition of the sensation, as distinguishing it from paraplegia arising from primary affection of the cord. In the thirteenth volume of the 'Med. Chir. Trans.,' he says: "Paraplegia dependent upon disease in the brain generally occurs about the middle or a more advanced period of life than is usual in diseases of the bodies of the vertebræ or the intervening fibro-cartilages; its progress is more rapid than the slow, insidious approach of symptoms from the latter diseases; the affection is more general, occasioning more or less paralysis of the upper and lower extremities; and this will often take place in a very few days from the occurrence of the complaint. This disease happens much more frequently in men than women; the gait of persons suffering from cerebral affection is peculiar and very different from that attendant on affections of the spine; it very nearly resembles the vacillating steps of a drunkard. Such paralytic persons are incapable of walking in a direct line; the limbs are loose, and thrown forward with an exertion of the whole body; there is a great consciousness of feebleness in walking, and the greatest difficulty in turning round. The appearance of the eyes often much resembles those of a drunkard, particularly when the patient is at all excited or anxious. The above similitude to the staggering steps of intoxication is readily understood, if we consider that it is the temporary disturbance of the brain from the congestion of its blood-vessels that deprives the drunkard of the power of directing his steps, and for the time induces a state bearing the closest resemblance to paraplegia."

"*Sensation is more impaired* than in spinal affections, when it will often remain perfect after a total loss of the locomotive powers. This impaired sensation is often peculiar, imparting an idea of some foreign body, as a leather glove or stocking, being interposed. The patient appears to feel, if I may use the expression, through a false medium. The limbs are more wasted and flabby, without any spasmodic rigidity of the muscles, which so often occurs in affections of the spine. Although often accompanied with a torpid state of the bowels, aggravated, no doubt, by the impaired muscular power of the abdominal parietes, there has not, in any instance that I have witnessed, been any train of gastric symptoms similar to those which so constantly attend affections of the spine, especially of the dorsal region."

We are not, at present, in a condition to determine what is the precise state of the nervous system arising from exhaustion of its energies. It is one which leaves no obvious changes behind it, although its results are often permanent. Losses in trade, reverses of fortune, depressing emotions, too much anxiety, too much study, and many similar causes, may so lessen the whole tone of the system as to produce anæsthesia with paraplegic weakness, and leave no change behind; or we find, as concomitants, sub-arachnoid and ventricular effusions, with wasting or other general alterations of the nervous substance.

Although it cannot be denied that a preponderating weakness of the lower extremities may arise from mere local affections in the encephalon, especially when affecting the parts in the neighbourhood of the spine, yet, as such cases follow the law of local central lesion, in which motion suffers more than sensation, already considered, I need not allude to them here, my object being to point out those cases which militate against this law, and to prove that they have an especial morbid anatomy, consisting, when any appearances are evident, of general and passive changes, of fullness of the larger venous trunks, watery effusions with chronic and various general states of wasting, hardening or softening of the nervous substance; changes which, though so seemingly opposite, may yet have the same result on the functions of the parts.

There is good reason to believe that many of these changes are *results rather than causes* of the loss or diminution of function. We know that we cannot interfere with the function of an organ without affecting its structure. If a duct be tied or obstructed, we get wasting or degeneration of the gland from which it comes, and I would venture to suggest whether something similar may not occur in the nervous tissue. These cases of paraplegia having an encephalic origin, and characterized during life by prevailing anæsthesia, present to us *general* as opposed to *local* and *isolated* changes of structure, that is provided there be any obvious appearances at all, which sometimes there are not.

The two following cases are from the abstract of an interesting paper on "Paraplegia," by Mr. Atholl Johnson, as I find it given in the 'Medico-Chirurgical Review,' 1842, and which are classed by him as under "Paraplegia, cause not discovered by dissection:"

Thomas B—, æt. 39, admitted into St. George's with paralysis of the lower extremities; motion is not entirely lost, but greatly impaired.

Sensation entirely lost at the soles of the feet, becoming less affected as you passed up the legs. Urine and fæces passed involuntarily. States that the attack commenced three weeks ago with numbness of the feet and dulness of vision in the right eye. At present vision is perfect, and the pupils act naturally. Sloughs on the back. Soon after admission, a cough with muco-purulent expectoration, mixed with blood, came on. In six weeks he died.

Post-mortem.—The body was much emaciated; large and extensive sloughs over both hips and sacrum. There was a small quantity of fluid in the upper part of the theca, which had apparently dropped down from between the membranes of the brain. Spinal cord apparently healthy; a slight alteration of colour and consistence was thought to be observed about opposite the first lumbar vertebra. The change, if any existed, was very slight. The sinuses of the brain contained a considerable quantity of dark blood; some fluid in the ventricles. Structure of the cerebrum and cerebellum apparently healthy.

Anne C—, æt. 21, admitted with palsy of the lower extremities; *sensation impaired, but not altogether lost;* double vision occasionally; incontinence of urine, but not of fæces; sloughing of back rapidly came on, but which, by the greatest care, was prevented from extending. Under the remedies employed she improved gradually for some time, becoming able to retain her urine and fæces, and to move the leg slightly when in a recumbent posture. The double vision still continued, though in a less degree; suddenly, however, the back and hips began again to slough, the legs were drawn up convulsively close to the abdomen, vision became affected to a much greater degree, and there was violent pain in the head. She died at last, worn out by the profuse discharge from the back. On examination after death, the substance of the cerebrum was firm and natural; it was, perhaps, rather wet when cut into. But little fluid in the ventricles. Substance of the anterior part of the middle lobe of the left hemisphere slightly softer than other parts. The cineritious substance was here of a pale colour; the cerebellum, pons, crura cerebri, and optic nerves apparently healthy. A small quantity of fluid was effused external to the dura mater in the spinal canal, and a very small quantity within the the theca; cord quite healthy.

Some time since I had the opportunity of seeing the following case of paraplegia, which arose, apparently, from mental emotion, and in which no lesion was discoverable after death. I cannot state what was the condition of the sensation.

Mary H—, æt. 21, of delicate frame but healthy, was in her usual health until one day a medical man, to whom she had applied on account of a bursal enlargement at the back of the wrist, offered to disperse it. To this, though very fearful, she consented. He struck the wrist several times with a book, and gave her so much pain, and mental disquiet, that she fainted. The same day she took to her bed, complaining of weakness in her legs. She soon became completely paraplegic, and her back sloughed. After lingering for some months she died, and, on a most careful post-mortem examination, no disease could be discovered in the brain nor spinal cord, nor any visceral derangement to account for the paraplegia.

It is in the experience of most to have seen cases of paraplegia in young women in whom post-mortem examination has not shown any local or general disease of the cord. In some of these congestive derangements of the kidneys have been found, with or without calculus in their pelves, and some have regarded these latter conditions, when present, as the exciting causes of the paraplegia, through impressions on the sympathetic nerves. It is not the object of this communication to enter upon such an inquiry, but I may express my belief that many of these renal affections in paraplegia are the result of the spinal affection. I do not here refer to those cases of kidney disease and stricture complicated with paraplegia, described by Mr. Stanley ('Med.-Chir. Trans.,' 1833), in which he believes that the diseased kidneys communicate an impression to the spinal cord and nerves issuing from it.

The following examples seem to belong to the class which might be termed *encephalic paraplegia,* which I am here considering, and illustrate the general symptoms presented by these cases, especially in reference to sensation.

Henry C—, æt. 53, was admitted into Guy's with the following history and symptoms:—He is tall and of a spare frame, large, well-formed head; attributes his present condition to losses in business and to irregularities, although of late he has been less dissipated. He has great weakness of memory. There is so much affection of the nerves of common sensation that in walking he does not seem to touch the ground, and the legs are so weak and faltering that it is with the greatest difficulty that he can support himself a few steps with a stick. He says he feels without weight, and finds it difficult to keep himself on the ground, for he seems to have a greater tendency to fly than to walk, though, indeed, he can effect the latter but a few steps only.

The hands and forearms are also affected with numbness, especially the former, and there is weakness and awkwardness of movement, but less than of the legs. He can retain or pass at will the urine and fæces, but there is some diminution of power; urine pale, large in quantity. The vision is impaired.

Heat and cold are oppressive. Twilight is more agreeable than full daylight, which is too exciting, and leaves him with less command over his movements. If he attempts to read, the letters soon become confused.

His memory is defective, his few weeks' residence in the hospital seeming to him a lifetime.

Various tonics, as zinc, iron, shower-bath, electricity, were employed without any good result, and he remains much in the same condition.

Sarah S—, æt. 30. In the autumn of 1845, whilst residing in Italy, where she had been for five years, began to complain of languor and indisposition to exertion, which she attributes to sedentary habits.

The attack began by a feeling of numbness in the left leg and right arm; also the vision of the left eye was impaired. In a short time the other extremities became similarly affected. At this time she could walk and grasp firmly, but could only imperfectly feel the floor, or tell when she held anything in her hand. After her return to England, which was in the spring following the autumn in which she was attacked, she became much worse, and was confined to her bed for five months. Galvanism in the course of the lower limbs restored her. She was then obliged to gain her livelihood by close application to her needle, and her present state is very unfavourable. I have watched her for a year. About two months since (two years from beginning of attack) her speech became faltering, and her eyesight very dim. The right arm and left leg are the worst, the left arm being not greatly affected; still there is some numbness in the fingers, especially in the tips of the middle and ring finger.

In attempting to walk, which she can do with slowness and caution, she has no sensation of touching the ground, but seems to tread on nothing. There is considerable impairment of sensation up to the knee, as if some soft and thick substance was covering the surface; above the knees the sensation is good.

The middle and ring fingers of each hand are more numb than the others, and the palm more than the back of the hand; the numbness extends to the elbows. She has power over the bladder and rectum. The various remedies employed have not had any very decided effect. They have consisted of tonics and rest, with counter-irritants.

The peculiar state of the sensation in this form of paraplegia was remarked, as I have said above, by Mr. Earle. There is numbness and an affection of the nerves of common sensation, as if some substance interposed itself between the object and the skin, and the integuments feel as if bandaged. The paraplegic affection is in part owing to the affection of the sensation, so that the patient cannot direct the muscles, rather than to an actual weakness in their contraction. One patient told me he could not walk without looking at his feet, because he felt as if the legs were cut off below the knees, and another because he had no apparent weight. With the numbness there is yet the perception of pricking or pinching of the integuments, and the numbness generally terminates in an undefined manner about the elbows, and just above or at the knees. Power over the bladder and rectum is not lost, except in extreme cases.

There is often pain over the head, some affection of vision, as dimness or muscæ volitantes, noises in the ear; and one peculiarity is that the most moderate pressure on the nerves, as from lying on the arm in bed, or sitting on the edge of a chair, will very speedily render the nerve completely anæsthetic. In walking the patients complain of difficulty of keeping on the ground, which seems due to want of sensation, which is necessary for directing the movement, as well as to the sudden contraction of the muscles. The morbid changes I have spoken of above are *general* rather than *local*, and, so far as I can find, afford no explanation of the peculiarity of the state of the sensation, although it seems probable such may depend upon disorder in or about the mesencephalon.

LECTURE III.

CERVICAL PARAPLEGIA—HEMIPLEGIA.

In the former lecture three forms of paraplegia were enumerated, in each of which there exists a characteristic condition of the sensation; and cases were given which seemed to show, that when anæsthesia prevails over loss of power, we must not expect to find a local and circumscribed lesion of the cord, but that we should refer such cases to peripheral affections of the nervous expansion in the tissues or to a general though often inappreciable change of structure of the myelencephalon. Before leaving the subject of the former lecture, it seems necessary to call attention to a form of paraplegia particularly affecting the upper extremities. Five cases of this particular form of paraplegia have fallen under my observation, and several others are on record. The legs are sometimes not in any degree affected; in all they are much less so than the arms. The muscles of the shoulder and upper arm are first implicated, and often alone affected. The disease may begin in both extremities at the same time, or, having begun on one side, may gradually pass across and affect the other.

At the onset there may be pain and soreness of the muscles, but after the disease has existed for some time, there is no obvious modification of the sensation. The following examples will illustrate this affection.

In the year 1830, Dr. Darvall recorded cases in which he designated "a peculiar species of paralysis," and which, from the absence of any more distinctive term, and the relation to paraplegia, may be classified under cervical paraplegia.

The following is an abstract of Dr. Darvall's cases, after which I will add others :—

"A washerwoman, who had been accustomed to carry heavy weights upon her arms, had paralysis, which was confined to the muscles which raise the os humeri; there was great emaciation of the deltoid; she could move the limb only a few inches from the trunk, and this with great difficulty. Nevertheless she could bend the forearm upon the arm, and, to use her own expression, *could do anything under the elbow.* The hand had its full power. There was not the slightest symptom of affection of the head, nor, indeed, excepting this paralysis, did the patient appear to have the slightest ailment. The treatment instituted did no good, and she gradually lost the whole power of the upper extremities."

"A porter in a cornfactor's warehouse, accustomed to move large bags of oats, &c. Incapacity limited to muscles which raise the arm, gradually invading the whole limb, and ending in total paralysis of both extremities. No pain was experienced in either of these cases. The state of the sensation is not mentioned."

"A man accustomed to carry heavy weights complained of *severe pain in the left deltoid,* and afterwards great weakness, in which the muscle was much wasted. Considering the pain to be rheumatic, acupuncture was tried, by the insertion of three needles into different parts of the deltoid. The result was most satisfactory; the pain disappeared, and the muscle recovered its bulk."

"A locksmith had been suffering from severe pain in the right shoulder for some time, which was relieved by colchicum, but the deltoid remained exceedingly flabby. Electricity soon restored the power."

"Female. Left arm only affected. Loss of power beginning in the muscles of the shoulder, and gradually extending downwards for four months; but *she can do anything under the elbow.* Slight pain in the deltoid on pressure, or on moving the shoulder. Muscles of the arm much emaciated."

Fanny J—, æt. 28, admitted under Dr. Addison, February 9th, 1848; a milliner, of delicate frame, fair complexion, regular habits; has not been overworked; catamenia regular; health good, with the exception of occasional headaches, with considerable intervals of entire freedom. Five months ago she had one of her usual attacks, which continued two days, and after a week she had a general heaviness and weight in the head, with dizziness and giddiness. This was followed by rheumatic fever. The right arm soon became paralytic, then quickly afterwards the left, and lastly, there was weakness in the leg of the left side. *She entirely lost the use of the upper extremities:* they were not swollen nor very painful. After a short time her general symptoms of fever disappeared, with a great improvement in the affected leg, and some in the corresponding arm. The right arm remained perfectly motionless from the shoulder to the wrist; there was some power in flexing the fingers. Her head is quite clear; no affection of the muscles of the face nor of the nerves of special sense. The paralysed parts are bathed in profuse perspiration, and the right elbow is highly swollen.

Tongue clean; pulse 76; bowels regular.

During the time she was in the house, the leg considerably improved, but still remained to a certain degree weak. The arms were very powerless, especially the right; but the contrast between the muscles of the forearm and those of the shoulder was striking; she experienced now and then shooting pains in the arms, especially the left.

During the whole course of her case, she complained of pain about the head; but this was peculiar, and seemed to originate in the upper part of the neck. There were several opinions as to the cause of this peculiar paralysis, some regarding it as the result of cerebral exhaustion, others thinking it was apoplectic. Dr. Addison, under whose care she was, considered it spinal, and the test of galvanism, from fifty pair of plates, showed great excitability of the muscles of the least affected arm.

George R—, æt. 32; admitted into Guy's under the care of Dr. Babington, for paralytic weakness and great wasting of the muscles of both arms, especially of the deltoid, supra and infra spinatus, biceps, and triceps. He says that six weeks ago he began to feel pain and aching in the shoulders, the right being first affected. He had considerable pain between the shoulders, and in the left breast. Quickly following these symptoms there was considerable *wasting of the muscles, with accompanying soreness of them.* For a short time after his admission the legs were weak, but this lasted for a few days only. There is now no obvious affection of sensation, but he has had a slight and almost inappreciable sensation of numbness in one side of the face and about the soles of the feet, which has passed away. The muscles of the forearm are much stronger than those of the shoulder; the latter are greatly wasted. There are no cerebral symptoms, no history of rheumatism, nor any obvious cause to account for the affection. He walks about with his arms hanging uselessly by his side.

(This man, since the above report was made, has been on alternate days galvanised through the affected muscles, and is now rapidly improving.)

Frederick S—, æt. 27, was admitted under Dr. Babington into Guy's in the year 1847. His present affection, which is peculiar, began to show itself very gradually four years ago. He is of middle stature, and has been moderately robust, but now presents a *remarkable atrophy of all the muscles supplied by the brachial plexuses of both sides, and of the muscles of the back at the upper part.* His legs are unaffected, and so are the muscles of expression and deglutition. He is by trade a dyer of silks, and much exposed to vicissitudes of temperature; the arms in his occupation being uncovered, and alternately immersed in hot and cold liquors. The substances employed by him as dyer are chiefly iron and vegetable astringents. They do not seem to have affected his general health. There is no affection of sensation, no tenderness of the spine, nor any in the course of the nerves; no affection of the brain.

Digestion, secretion, and circulation normal. It is obvious in this case that the great inability to move the arms may be explained by their atrophied condition, and the disease is strictly a form of muscular atrophy and consequent weakness.

Dr. Bright, in his reports, gives the following case of "paraplegia connected with rheumatic gout:"

Daniel McC—, æt. 45, a very large and athletic man; a coal-shipper by occupation, and therefore greatly exposed to the vicissitudes of the weather. His general affection *is entirely confined to his hands and arms,* which are almost completely paralysed. His hands lie nearly powerless on his lap, and he has just power enough to raise his hands to his mouth, assisting himself by a kind of swinging motion of the body. No pain is experienced by rotation or nodding of the head, except a slight uneasiness *low down in the neck.* He has *rheumatic* pains in the feet. The affection of the hands and arms has been coming on gradually for eight months, and he says he was affected in a similar way three years before, but, after six months, recovered so much as to be able to return to his laborious employment. The back of the neck was blistered, and a few leeches applied daily to that part. He was ordered antim. opiate pill, with two grains of calomel twice a day, and occasional purgatives of colchicum wine and infusion of senna. However, very little improvement followed, and he continued the calomel until ptyalism was produced. Afterwards a fair trial was given to nux vomica, without any decidedly advantageous change. Still he seemed to gain ground, and expressed himself as being better. At length he became the subject of a most severe attack of acute rheumatism in the right arm and both the feet, which was treated by calomel, antimony, and opium. In a few weeks he left the house, free from all traces of his last attack, and so greatly relieved from his paralytic ailments as to intend returning to his laborious occupation.

The paralytic affection illustrated by these cases is peculiar. The muscles of the shoulder and elbow are much more affected than those of the forearm. The paralysis may be limited to the deltoid for a long time, and subsequently implicate other contiguous muscles. An arm, powerless in this particular affection, presents a remarkable contrast to the arm paralysed from apoplexy. In the former the forearm and the fingers are less affected than the parts above, whilst in the latter the reverse obtains. We have already seen that paralysis may arise from the centres, or from the nerves; but many of these cases here described show a primary *affection of the muscle,* others *both of the muscle and the nerve distributed to it:* in either case it is probable that the neurilemma forms the medium through which the diseased action travels to affect adjacent muscles. A man æt. 56, came under my notice for a wasting and a weakness of the muscles which elevate the humerus; the muscles of the forearm were of their normal size and strength. The account he gave was that, thirteen months previous, he had fallen and struck his shoulder, but the blow gave him no anxiety, and its immediate effects soon disappeared; but after a month there came on very gradually weakness of the deltoid and of the muscles about the shoulder, supra-spinatus, &c. After eight months the opposite side began to be similarly affected; *below the elbow the arms were quite healthy.* There were no spinal or cerebral symptoms. The head was clear, the legs strong. Dr. Darwall's cases are of a similar kind, and probably arose from the injury to the muscle by carrying heavy weights on the shoulder. We have also given above examples of this muscular atrophy arising from cold and rheumatism, and one in which there was no obvious cause. Although I cannot venture to give an account of its exact pathology, yet may I express my belief that the muscular tissue is the primary seat of this disease. I have now a case under my care in which I have been able to trace a continuous extension of the disease through the deltoid, beginning at its posterior edge. Soreness of the affected muscles is an early symptom; this, however, varies much, and is sometimes absent. The results of treatment also favour the same view. If taken early they seem very amenable to treatment, and especially to electricity. Darwall found acupuncture of the muscle arrest the disease. That paralysis may arise from primary affection of the motor organ or muscle, there seems no reason to deny; and it is more than probable that, in considering the causes of paralysis, such a source has often been overlooked. Muscle, even during rest, is, to a certain extent, active, and its due nutrition and susceptibility to excitement are, to a great extent, dependent upon this action, which is kept up by the connection of the muscle with the centre through the nerve supplied to it. Muscles rapidly waste when the nervous trunk which runs to them is diseased, and irreparable atrophy of muscle may result from causes of a temporary kind acting upon the nerve; hence the importance, in all cases where the muscular tone is interfered with, of supplying the stimulus of electricity to prevent the wasting which would otherwise follow upon the paralysis; for, if we do not employ this means, we may find, to our dismay, that, although the nerve may be restored to its functions, it may have no fibre to excite. Some time since I met with an excellent illustration of this principle in the case of a woman admitted under the care of Dr. Addison. She had had paralysis of the seventh nerve for many years; the eye of the same side was permanently open, and the cheek flabby and fallen. She never had paralysis of any other part; had been subject to faceache from bad teeth, and attributed the paralysis to this. There had never been any discharge from the ear, nor any obvious cause of injury to the nerve. She died from affection of the chest, for which she was admitted.

Post-mortem.—The medulla oblongata and cerebellum were removed with care, but no disease of either could be detected. The seventh nerve of the affected side was traced from its origin to its distribution, through the whole of its osseous course. It was not apparently wasted, and when examined by the microscope its tubules presented the usual appearance, and could be traced continuously. The muscles of the cheek were not distinguishable; no muscular fibre could be detected therein.

HEMIPLEGIA.

In my first lecture I spoke of the extent of the decussating influence of the hemispheres on the spinal cord, and mentioned what has been also remarked upon by others, that as the same law obtained equally for motion as for sensation, and as the number of fibres actually decussating was very small in comparison with the whole spinal fibres, we must regard those which did cross as commissural between brain and the segments of the cord, rather than as continuations of the nerves themselves. In applying this to the explanation of hemiplegia, it may be stated (although there are exceptions, not yet accounted for) that if there be disorganisation of the hemisphere, whether affecting the anterior or posterior lobe, and implicating the optic thalamus or corpus striatum, singly or equally, if the lesion be of such an extent as to produce hemiplegia, *there will be a greater affection of the upper than of the lower extremity, and a greater loss of motion than of sensation.* If the lesion has been sudden, and both the upper and lower extremities are completely paralysed, the leg will begin to improve before the arm, and the muscles nearest the trunk will regain their power first,—that is, the deltoid and flexors of the elbow before the muscles which move the fingers. This is so obvious and so constant that it may, I think, be laid down as the law of hemiplegic paralysis having the encephalic origin I have spoken of. As regards sensation, it is sometimes abolished, as the intelligence also is for a few days following the attack, if the lesion has been sudden, but is soon recovered from; the mode of its restoration being, like the recovery of muscular power, *first in parts nearest the trunk*, and first in the leg.

The seventh nerve is, in the class of simple cases, less affected than any nerve of the hemiplegic side; still it is generally to a slight degree paralysed, and the affection shows that it is subject to the law of crossed action; and the same may be said of the lingual nerve.

I mentioned in my first lecture that the third nerve was an exception, and probably the only exception, to the law of decussating effects; and an attempt was made to show why this was, and how the exception seemed to point out the use of a decussation. I must now trouble you with some facts in proof of my position.

In hemiplegia depending upon lesion of the hemisphere affecting the corpus striata and thalamus opticus the third nerve frequently does not suffer at all, the movements of the eye being unaffected and the pupils acting equally. In looking over the reports of a great number of hemiplegic cases one is struck with the slight affection of the third nerve, so that it requires a great extent of research and observation to find examples illustrative of the point in question.

It is now well established that the contractile power of the iris depends upon the third nerve, and equally certain that the act of contraction is excito-motor, and therefore in part governed by the state of the optic nerves. In studying the condition of the third nerve, therefore, it is necessary to have regard to the susceptibility of the retina. The following cases are given to show that the third nerve is affected on the side on which the cerebral lesion exists:

Richard K—, æt. 66. Sanguineous apoplexy ingravescent; comatose; breathing 26; pulse 64; entire paralytic resolution of the left side; pupil dilated on the side opposite to the paralysis, that is on the side of the cerebral lesion.

A. B—, æt. 67. November 29th, 1846, symptoms of ingravescent apoplexy. On my first seeing him his right pupil was very much dilated, left small, as in sleep; breathing 32, pulse 74; complete insensibility. It was difficult, from the condition of the muscles, to tell which side was most affected. *Post-mortem.*—Very large effusion into right hemisphere, probably beginning near substantia perforata lateralis.

Paraplegia. Catherine F—, æt. 56. Paralysis of left side, coming on in the course of two days, without loss of consciousness. Entire paralysis of arm and leg, and falling of the features on the same side. The opposite pupil (right) slightly the largest. She turns the eyes with greatest readiness to right side.

Hemiplegia with symptoms of ramollissement. Female, æt. 56. Pupils equal, and act on the stimulus of light; complete resolution of left side; eyes directed to right.

Acute hydrencephaloid affection. D. W—, æt. 19. Partial consciousness;

breathing rather stertorous, 20; pulse 120; left side of body appears paralysed; eyes directed to the right, jactitating motion of this side, right pupil largest, vision lost.

Hemiplegic weakness coming on during sleep. Margaret N—, æt. 28. Partial paralysis of left side, most affecting the arm; the sight of left eye impaired, and the pupil of this eye rather the largest; the motions of the eye are nearly perfect, *slight ptosis of right eyelid.* In this case the state of the pupil is the reverse of that in the apoplectic cases, depending upon the condition of the retina, but the partial ptosis in the side opposite the hemiplegia proves the law.

Hemiplegia, ten months' standing, attack sudden. Richard L—. Hemiplegia of right side, gradual improvement; turns his eyes most readily to left.

Chronic brain disease. W. M—, æt. 27. Hemiplegia, weakness of right side, with some anæsthesia, pupils both dilated, left least active.

These cases, which I might add to, will be sufficient to prove the rule I wish to establish. The investigation is difficult from the reaction, as I have before said, of the retina and the iris, and the uncomplicated examples are not common. The facts may, so far as I have observed, be reduced to:

1. Pupil largest on side of disease, vision being lost.
2. Eyes turned from paralysed side.
3. Ptosis on side opposite to paralysis of extremities and face.
4. Though no obvious affection of iris or recti, yet a patient may turn his eyes most readily from the affected side, and open the eye widest on the side of the paralysis.

I must take leave to remark, that it has seemed to me of the greatest importance to lay down with all possible precision the law of hemiplegia, inasmuch as, being thus a starting-point, we can reduce the apparently anomalous cases to their proper laws. Nor do I think we should have remained so long in a state of uncertainty upon the diagnosis of nervous affections if we had not taken it for granted that their whole nature was to be anomalous. There must be as invariable a law for nervous action as for any other physical phenomena; and, if anomalous, it must be because our law has not been fairly deduced.

Whilst I have been thus attempting to prove the certainty and definite nature of the law of common hemiplegia, a host of cases must have occurred to every one, which seem to be exceptions to the rule. My next attempt must, therefore, be to show that they belong to quite another law:

1. The leg is often more affected than the arm.
2. The seventh may be alone paralysed.
3. The third nerve is often alone, or to an extreme degree affected, and on the same side as the seventh.
4. The loss of sensation is often very much greater than the loss of motion.

CERVICAL PARAPLEGIA—HEMIPLEGIA.

Amongst the exceptions to the ordinary law of hemiplegia enunciated above are forced to be considered those in which the leg is more affected than the arm, or in which the former, contrary to what is usual, does not recover so rapidly as the latter. Although I feel that I am not at present in a condition to explain all these exceptions, yet so many of them may be explained, and the conditions here, as elsewhere, must stand to each other in such an invariable relation, that it may be hoped that such examples as now place themselves conspicuously against our generalisation may serve as landmarks pointing to a yet higher law.

Having once obtained a general expression for a class of facts, the mind becomes alive to such as do not run parallel, and seeks for modifying circumstances. Thus, having seen what is the general condition in hemiplegia, depending upon local lesion of the encephalon, if we find cases which do not present corresponding phenomena, we cannot refer them to the same cause. Exceptional cases to the law of local lesion may depend upon hysteria, or an allied condition; also upon complication of encephalic disease with disease of the cord; or arise from disease situated in the muscles or nerves of the paralysed member itself. I have also seen two cases of hemiplegia in which the arm recovered before the leg, in which post-mortem examination showed the disease to be at the inferior part of the posterior lobe of the cerebrum, neither the corpus striatum nor thalamus opticus being implicated. When the seventh nerve is alone affected, the causes producing the paralysis are for the most part so situated as to implicate the nerve in its course, or at its immediate origin.

I have not seen or been able to find the records of any cases which form exceptions to this. The complicated course of the seventh nerve through the bony canals of the petrous portion of the temporal bone, and subsequently its exposed position on the face, render it very obvious to disease.

Amongst the exceptions to the ordinary law of hemiplegia none are more common than those of the third nerve. In hemispheric lesions of a moderate extent this nerve is rarely much affected, yet it is amongst the nerves of the body most subject to paralysis.

Before detailing the general phenomena of these cases I will call your attention to the origin and intercranial course and communication of the third pair of nerves, which have also been so ably remarked upon by my friend Mr. John France, to whom we are indebted for the fullest report of a large number of cases of paralysis of the third nerve.

The nerve has its origin near an extremely vascular portion of the brain, the substantia perforata postica; to use Mr. France's own words, "we find the nerve, almost throughout its intercranial tract, in the immediate vicinity of those which must be regarded as very dangerous allies; first hooking round the posterior cerebral artery to traverse the narrow interval between that vessel and the superior cerebellar, then running forward nearly parallel to the posterior communicating artery, then crossing the termination of the internal carotid immediately on its outer side, and closer to it than any other nerves in the cavernous sinus."

"The sixth nerve, it is true, is previously in actual contact with the coats of this vessel, but running along the floor of the sinus, must in a great degree be secured from pressure, as from the upward direction of the current of the blood the horizontal portion of the carotid must be rather raised from than pressed against the inferior wall of the sinus upon each contraction of the left ventricle, and from the same cause the inferior wall of the artery itself must be mechanically the least liable to morbid distension or rupture; that nerve, however, sometimes suffers like the third."

But not only has the third nerve these dangerous allies in its course, but, from its position and the gravitating tendency of subarachnoid effusions, it is most subject to be pressed upon in its course by inflammatory effusion.

Also, near its entrance into the orbit, it is joined by branches of the sympathetic from the cavernous ganglion, and by a branch of the fifth, which extensive nervous communication explains the functional or sympathetic affections of this nerve.

Double vision is a common effect of paralysis of the third nerve, which, if unexplained, might lead to the supposition that the central sensory ganglia were implicated; but in these cases the diplopia arises from the want of consensual action of the recti muscles of the two eyes, so that they cannot be brought to adapt themselves to the same object; and hence, as Mr. France has remarked, we get two forms of diplopia, "one arising *from disagreement of the planes* of the optic axes, as when an object is held towards the faulty side, but above or below the level of the eyes, one only of which can be raised or depressed; the other *from defect of convergence of the axes*, as when an object is presented on the level of the eyes, but on the sound side, towards which the affected eye cannot follow it."

In many cases of paralytic affection of the third, as shown by ptosis of one of the eyelids, we cannot suppose that any central organic disease exists. The class of patients in which they occur, and the rapidity of the cure, the nature of the means which are known from experience to be most successful, together with the complete localisation of the affection, justify this opinion.

The cases of ptosis recorded by Mr. France, in the Guy's Hospital Reports, illustrate the general history of these cases very well. The remarks given above, upon the intimate connection between this nerve and the large venous sinus and the arterial trunks, together with the the tendency of membranous inflammation to occur at the base of the brain, and for effusions to gravitate there, will serve to elucidate much of the obscurity of their pathology. I shall here quote two or three cases given by Mr. France:

George D—, æt. 12, who had in infancy suffered from hydrocephalus, and possessed a somewhat disproportionately large head, presented himself at the Eye Infirmary of Guy's Hospital on February 7th, 1845. He was subject to frequent attacks of pain in the head, particularly just over the left eye, and sometimes at the corresponding point on the opposite side, and to frightful dreams, disturbing his sleep, and causing him to scream violently. When most troubled with headache, often two or three times a week, he had sickness and vomiting, apparently unconnected with errors in diet. His general aspect was indicative of debility; he was sallow, but in a manner intelligent and lively. Nine days before application he was seized with headache and vomiting, which lasted together with frequent twitching of the left eyelid, for a couple of days, when the lid dropped, and he became unable to elevate it without the assistance of the hand. Upon the patient's application, ptosis was complete, but he could slightly raise the lid on wrinkling the forehead by the action of the occipito-frontalis. Abduction of the affected eye was well performed; the eye then slowly returning towards, but not quite reaching the centre. Adduction, elevation, and depression, more especially the two latter, were impracticable. The pupil, examined separately, was active, but inclined to remain rather more dilated in the dusk than the opposite pupil. The sight of either eye was good when both were employed; double vision resulted, unless the objects were held to the temporal side of left eye, so as to permit the axes to correspond.

The boy was put under the influence of mercury sufficiently to redden the gums, and repeatedly blistered, and was discharged with restored power over the globe and palpebra, after about seven weeks.

Henry H—, æt. 26, a fishmonger, subject to occasional bilious derangement, and, in hot weather, to pain across the forehead, for the relief of which he had habitually employed leeches with benefit. He was, six weeks before application at Guy's, suddenly seized, immediately after a walk of six miles, with severe pain in the head, tinnitus, vertigo, and sickness. He had not partaken of anything likely to disorder the stomach. The sickness, however, accompanied with the other cerebral symptoms just mentioned, and with loss of power of elevating the upper lid on the right side (which took place suddenly on the first onset of these symptoms), continued for three or four days. He was twice cupped, was blistered at the nape of the neck, and had purgative medicine administered; and under these remedies the sickness subsided, and the power of the levator palpebræ was in some degree restored. On application at the hospital, November 10th, 1845, he still complained of headache and vertigo; the right upper eyelid could only be raised so far as to uncover about a third of the pupil, unless the occipito-frontalis was called into action; the movements of elevation, depression, and adduction of the globe were much limited, and the globe when quiescent maintained the position of abduction. There was diplopia in regarding any object to the left of the median line, and the right pupil, which the patient spontaneously described to have been at first much larger than the left, was still a trifle more dilated and sluggish than the latter, which was of medium size and active. He was ordered compound calomel pill, and two grains sulphate of zinc with the compound infusion of roses and salts, three times daily, and a blister to the back of the neck.

November 21st.—Though convalescing, the patient still complained of occasional vertigo and headache in the morning; he could raise the right nearly as well as the left superior palpebra, the motions of the globe were almost perfectly restored; horizontal diplopia had ceased, there yet remained a trifling increase in size and indolence in action of the pupil. A grain of quinine was added to each dose of his mixture, two of the daily pills having been previously discontinued.

28th.—He was nearly well. A just perceptible difference existed between the degree of elevation of the two superior palpebræ; the power of adducting the right globe was perfect; that of elevating and depressing it still restricted within less than the natural boundaries; hence, double vision which had ceased a week before in the horizontal direction, was still manifested at the extremes of the perpendicular movements of the eyes. Some indolence was the only remaining morbid affection of the pupils.

John C—, æt. 28, a gas-work labourer, usually in the enjoyment of excellent health, who had abstained from indulgence in liquor, and been exempt from exposure to the furnace for some time before his application at the hospital, September 29th, 1845, was attacked about two months previously with cephalalgia, vertigo, and double vision, which were not preceded by any irregularity in diet, and subsided without treatment in a couple of days. For eight or nine days before he applied at Guy's he suffered from severe pain at the occiput, extending along the right side of the head, occurring chiefly in the night and on his rising from bed in the morning, and lasting for two or three hours at a time. This was followed by vertigo and diplopia, and shortly after by dropping of the right upper eyelid.

When he came for advice he was free from pain, and unconscious of any other ailment than complete inability to raise the lid, though he could slightly separate it from the inferior palpebra by wrinkling the forehead by the occipito-frontal muscle. The position of the globe, when at rest, was with the cornea directed straight forwards. The patient could abduct the globe freely, and adduct it too, as far as the centre; he could also elevate and depress the globe, but within very narrow limits only, whence objects held at all to the left side of the median line, or much below or above the level of the eye, were perceived as double. The pupil of the right eye was of thrice the diameter of the left, but acted conjointly with it; and also, to a slight extent, independently: the left eye was unaffected, and no other paralysis existed. He was ordered cupping to ten ounces, and a purgative at once—two grains of calomel with one of opium three times daily; chalk mixture if necessary, and the stronger mercurial ointment for inunction on the temple.

Under this treatment he rapidly improved. At the expiration of a week he could raise the lid so as to expose three fourths of the pupil; he could adduct the cornea considerably past the central point, and raise and depress it more extensively. Double vision of objects on the left continued, and the pupil was equally dilated, but perhaps more active. The mouth was becoming sore. In another week the motions of the lid and eye were almost entirely recovered; the pupil was reduced to two thirds of its late habitual size, and had regained its briskness of movement, and the patient shortly after discontinued his attendance at the infirmary.

A very rare cause of paralysis of the third may be aneurism of one of the arteries lying near it in its intercranial course. Such a case occurred under my own observation, and is recorded also by Mr. France. The following is an abstract of the case: Sarah S—, æt. 20, a stout and short-necked girl, of a plethoric habit, and subject to constipation, stated that for a month she had suffered from headache and giddiness whilst sitting at needlework, and that five days before admission she was suddenly seized with pain over the right eyebrow, faintness, and vomiting. Leeches were applied with relief, but the next morning the right upper lid was dropped, and vision of the same side impaired. She had, on admission, been bled, leeched, and purged, notwithstanding which the fall of the lid and impairment of vision had both increased. Seven days after coming into the hospital, she retired to bed complaining of headache, from which she had not, while in the hospital, been quite free. She slept comfortably until five o'clock in the morning, when the nurse's attention was attracted by her moaning. She was then found in a state of insensibility, without convulsion, deep low stertor, and froth at the mouth and nose.

On a post-mortem examination there was found considerable recent extravasation of blood at the base of the brain, penetrating extensively into the meshes of the pia mater between the convolutions. The hæmorrhage had taken place from the bursting of a small aneurism situated on the posterior communicating artery of the right side, and which had produced compression of the right third nerve.

The communications of the third nerve with the fifth and sympathetic, may explain the apparently functional character of many of its affections. Many persons in whom they occur are weak and debilitated subjects, with obvious disorder of the general health, and are relived by alteratives, purgatives, and tonics. M. Marchal has given, in the 'Archives générales' (tome xi), a memoir on paralysis of the third pair of nerves following upon neuralgia of the fifth pair. He believes that in many cases the lesion of the third is subsequent to a retrograde action, propagating itself from a few filaments of the fifth, and he gives his opinion that the course of such influence is through the lenticular ganglion.

It is well known that irritations of the fifth from diseased teeth, or a blow, may cause amaurosis, but yet it may be doubtful whether the cases recorded by Marchal in support of his opinion be correct; whether the anæsthesia of the fifth nerve, and the supervening paralysis of the third, are not results of a common cause, having its seat in the membranes, and affecting these nerves in their intercranial course. It is sufficient, however, for the main argument of this lecture that they do not invalidate the law of hemiplegia, inasmuch as they belong to an entirely different series of disorders. Whether membranous or sympathetic, I have not now the time to discuss, but they have certainly not a hemispheric origin.

The most remarkable exceptions to the law of hemispheric lesion present themselves to us in the affection of the nerves of sensation, and I particularly direct attention thereto.

First.—Because they bear upon the question of the existence of separate centres in the brain for common sensation and voluntary motion.

Secondly.—In reference to the pathology of these cases; because I think one class of them, at least, is strictly functional.

Without entering upon a discussion of the seat of sensation, or where the sensorium commune is situated, I would call attention to several conditions under which anæsthesia occurs.

First.—Anæsthesia is produced by diseases of the blood. Ether, chloroform, Bright's disease, and gout supply us with examples.

Secondly.—Anæsthesia is said to arise from mental states, as in mesmerism. It certainly may be produced by nervous exhaustion, by a shock, and often exists in hysteria.

(*a*) It accompanies the shock of apoplexy, but is generally not persistent. (*b*) It may form a part of the phenomena of epilepsy. (*c*) It may follow venereal excesses, over-study, and liver derangements.

Thirdly.—Anæsthesia forms a prominent symptom in one form of paraplegia.

Fourthly.—Anæsthesia may be an affection of the peripheral nerves.

I know of no cases of anæsthesia unaccompanied by loss of motion which arise from local disease of the encephalic centres.

My first assertion that anæsthesia may be the result of blood diseases, requires no very difficult train of facts to substantiate it.

My attention was first called to the peculiarity of these cases by the following. The error I then made in diagnosis has been of the greatest service to me as pointing to a different pathology of these cases from that which would refer them to local central lesion.

In April, 1843, a male patient, æt. 35, was admitted under Dr. Addison into Guy's with the following history and symptoms:—He is by trade a painter, lives but poorly, and says he drinks nothing but water. In the pursuit of his vocation he is compelled to take long journeys, and to remain for many hours standing to paint. Yesterday morning (the report says), when at work, he fell from the steps on which he was standing, quite insensible to the ground, but he soon recovered his consciousness, and found that he had complete anæsthesia in the right half of his body, including the integuments of the forehead and face.

After being in the hospital a short time he was found in a drowsy state, and when roused seemed confused. The pupils were natural. There was some muscular weakness and awkwardness in the right side. He has a very vacant look. Pulse 75. Hypertrophy of left ventricle; urine highly coagulable; sp. gr. 1018; small in quantity. During the course of five weeks the sensation returned to the parts affected, but with fluctuations About the 18th of June, that is two months from the attack, he was suddenly seized with all the severe symptoms of renal catarrh with pleurisy, and died the next day.

Dr. Addison, under whose care, as I have said, the case occurred, had prepared me for a disappointment in my anticipations of local lesion; yet, from the suddenness of the attack, and the complete hemiplegic character of the malady, I must confess I should have been a disbeliever had we had no post-mortem examination. I wrote in my note-book at the time, a series of reasons why there should be, and why there should not be, any local disease.

The brain was anæmic; no lesion, nor traces of one, could be detected, although it was carefully sliced throughout. The tissue was of the normal consistence. No local ramollissement nor effusion existed. The heart was hypertrophied. Aorta healthy; large vessels good. Kidneys contracted, granular, mottled, firm, with some serous cysts on the surface.

Modern discovery has supplied us with abundant proof of the anæsthesic effects of blood diseases in the phenomena produced by the inhalation of ether and chloroform. Nor are we, I think, surprised that a patient should be able to move his limbs, to converse and direct his eyes to various objects, to be to a considerable extent conscious; and yet that he should be quite unaware of the operations of the surgeon on the extremities at this same time: when we take into consideration the extreme extent of nervous distribution in the tissues, that the ether is permeating all these, and that the skin, from its rich supply, must be largely affected: whilst, as I have often seen by experiment on this subject, muscular contractility is not affected by the circulation of ether, nor even by immersion in it. It is however probable, that there is, with these diseased conditions of the blood, some modification of nutrition.

The second class of cases in which anæsthesia preponderates over loss of motion are probably functional, I mean *essentially nervous, restricted to the nerves*, not caused by a hyperæmia, nor a local determination of blood, nor a congestion of the blood-vessels, nor by impurity of the blood.

A nerve has its aptitude to perform its function in virtue of its proper organisation, as muscle is contractile as the result of its physical constitution.

A nerve in action, is probably undergoing a more rapid change in its organisation than a passive nerve; but, independent of this, we must admit, that there is strictly functional activity; for, whilst a nerve conducts the mandates of the will, or receives impressions from without, it is in action through all its length and breadth, yet we cannot admit that its entire organisation is changed. Perhaps the conducting wire of a battery may serve to convey my meaning. When it connects the two ends of the series, it is still a copper wire, but it has a new function—it is magnetic. The current does not change it organically, though it does functionally; yet the current is apt, in time, to affect its organisation. Thus the heat induced, renders it more exposed to oxidation; and, if the wire be of a substance quickly to oxidate, its consequent organic change would be quicker. But, without taking up your time with such speculations, it will, I think, be admitted, that the present class of cases are functional, and that to view them in any other way is apt to lead to dangerous practice.

About two years since, I was one evening called in great haste to a gentleman well known to me. He is of thin, spare frame, about five feet six inches in height, active and very excitable, nervous, has a multiplicity of duties to perform, and no repose in his disposition. It was about half-past seven in the evening, he told me, in great agitation, that he had lost the use of his right leg and arm, but on investigation I found he could forcibly throw forward the leg or the arm, but there was an awkwardness in directing their movements; the most prominent feature in his case being the anæsthesia; the awkwardness seeming to be the result of diminished sensation, so that he could not tell the whereabouts of the limb, rather than to arise from loss of power. This being totally opposed to the law of hemiplegia, I was disposed to regard it as functional, the result of nervous exhaustion, and prescribed nothing but a long and sound sleep. The following morning he was well.

A gentleman, recently married and overwrought by mental exercise, complained to me that he had just been seized with numbness of the right leg, and that on attempting to walk he found his gait very awkward, and he feared he was going to have an attack of paralysis. On inquiring, I found that the want of power over the muscles arose from the numbness, rather than from any absolute want of muscular power, and was an awkwardness rather than paralysis. Using as we do our muscles under the direction of sensation, this will be readily understood. Knowing his general condition, I advised vacuity, sleep, and a tonic, and all his symptoms soon disappeared.

It would occupy more than the whole time allotted to these lectures to point out the strange vagaries of sensation manifested in these cases, and the apparent paralyses that accompany them. The object I have is to show that *where the loss of sensation and its various modifications are so great, with much affection of the muscular power*, and often in no other way than I have described above, *the disease does not arise from central lesion of a local kind*, and that these cases are not exceptions to the law of hemiplegia already defined, but come under quite a distinct one. The remedies approved of by experience warrant such a conclusion, such as *vacuity from business, sleep,* mild nourishment, moderate purgation, and good air and exercise. *Congestion* may occur, but never admits more than gentle local depletion.

I have known one case in which the strange vagaries of sensation would not let the patient sleep, for just as consciousness was lost a strange creeping came over the scalp and woke him; this was so continued, and so certain, as at last to reduce the patient sadly. It was obviated by his wife watching him, and as he fell off she gently passed her hand over the head, and the pressure so produced on the nerves drove away the subjective sensations, and he slept.

Of the state induced by mental impressions, or from nervous exhaustion, induced by monotonous movements or otherwise, called mesmeric, I have no experience, and cannot, therefore, give any opinion thereon.

I would, however, place in this category of anæsthetic affections those strange cases of apoplexy in which there is paralysis of one side of the body and anæsthesia of the other, the paralysis depending upon lesion of the hemisphere, and following the law of hemiplegic paralysis, the anæsthesia occurring without any lesion in the hemisphere which corresponds to these parts, and is functional after the same manner that we get convulsions on the side which is not paralysed, the result of the shock of the injury on the other hemisphere. We cannot, therefore, regard it as the effect of a local lesion, nor an exception to the hemiplegic law, nor any evidence of separate centres for sensation and motion.

A tingling, a transient numbness, a feeling of coldness, a creeping, or something equally trifling and transient may precede severe and often sudden disorganising processes, and yet, strange to say, as the well-marked paralytic effects are produced by the lesion, the affection of the sensation shall fade away. They have been like the cloud before the storm—it is now passed; the oak is riven, but the sun still shines. These anæsthetic phenomena are thus the signs of deranged function, which, we know, often precede greater changes. Abercrombie gives the case of a young lady, who for two or three years suffered from transient hemiplegic anæsthesia; this functional disturbance foreshadowing the ramollissement which, at the end of this time, came on. There are on record many cases, said to be anomalous, in which the sensation of parts has been much diminished or permanently lost.

I quoted some of these in my last in speaking of peripheral paraplegia, in which the morbid impression seemed to have commenced in the integuments from cold, or from some inexplicable cause, as in those remarkable examples occurring during the epidemic in Paris. I think if the cases, whose general details are shown by the following examples, be viewed in connection with those of peripheral paraplegia, we shall not find them anomalous, but have to refer them to the cases of peripheral disease.

Dr. Yelloly, in the third volume of 'Medico-Chirurgical Transactions,' gives a case of anæsthesia which strikingly exemplifies this view. It occurred in a man aged fifty-eight, a Scotchman, after being much fatigued and heated in his attendance as one of the grand jury in Kingston, in Jamaica, in very sultry weather, he went to bed with the window open. On awakening in the morning he found his feet and ankles perfectly numb, but without pain, and without the muscular power being at all affected. Soon afterwards he felt a numbness, with a tingling pain in his little finger, such as occurs in a part asleep, and by degrees finger after finger became affected until both hands were to a great degree insensible. No head symptoms, nor any affection of his general health nor viscera. A physician of eminence attributed the disorder to scurvy; but he had no cuticular affection until twelve months after the occurrence of the numbness, when some red pimples showed themselves on his legs on his lying down on the ground after walking up a steep hill. The hands up to the wrist, and the feet half-way up the leg, were perfectly insensible to any species of injury, as cutting, pinching, or burning; the insensibility did not suddenly terminate, but it existed to a certain degree nearly up to the elbow, and for some distance above the knee.

His hands were of a somewhat purple hue. If he wished to ascertain the temperature of anything, he was under the necessity of putting it to his face or neck, or upper part of his arm. His skin seemed to be unusually affected by heat. His hands were never free from blisters, which he could get by inadvertently placing them too near the fire.

The power of motion existed in the muscles of both hands and feet. He could grasp pretty firmly, but in holding anything he was apt to drop it if his attention was at all called away. The susceptibility of impressions generally, as well as the muscular power, seemed to be diminished.

The result of the case is no further given than that the patient was not benefited by the treatment. He left England again for the West Indies. I cannot but regard this as the result of injury to the cutaneous nerves, arising, in the first instance, from exposure whilst heated.

In enumerating the apparent inconsistencies of anæsthesia, Abercrombie says that a gentleman who was under the care of the late Dr. Hey, of Edinburgh, had two paralytic attacks at the distance of eight months from each other. In the first there was perfect loss of feeling with only partial loss of motion; in the second there was perfect loss of motion with only partial loss of feeling. He recovered perfectly from the first attack after a short time; but after the second, though he recovered partially, he continued to drag his leg, and, after a year or more, died of apoplexy. The different results of the two attacks precisely agree with the principle I have been endeavouring to establish. I believe there is yet a source of many functional diseases of the nervous centres which, in modern medicine, has been too much neglected. I refer to the interweaving of the sympathetic with the nerves, and, as we have reason to believe, entering into connection with the nervous centres themselves. States of the brain produce functional disorders of the stomach and liver, and these in their turn produce strange nervous conditions; amongst the most important of which to be here named are numbness of the fingers or face, pain over the eyebrow and on the occiput, partial paralysis of the retina, giving rise to hemiopia or indistinctness of vision, with vertigo, all of which often vanish at once by good exercise or an active purgative. Although this attempt to generalise and classify nervous affections is necessarily very defective, yet should it hereafter be found that a careful attention to the degree in which sensation is affected in relation to paralysis may serve to inform us whether the disease be local or general, functional or organic, as indeed I think it will, the object of these lectures will have been attained.

CASES OF PARAPLEGIA.[1]

PARAPLEGIA FROM TUMOURS COMPRESSING THE CORD.

TUMOURS growing in the cord, or from its membranes, are among the more rare causes of paraplegia. With the exception of scrofulous deposits, these formations are most frequently seated in the loose tissue under the visceral layer of the arachnoid, or grow from the inner surface of the dura mater. They have generally been regarded as malignant, but their microscopical characters, their indisposition to invade or infiltrate surrounding parts, and their non-occurrence simultaneously in other organs, refer them to a simpler class of tumours, the *fibro-nuclear* or *fibro-cellular*. The cord and its membranes appear to be extremely rarely affected primarily by cancerous growths. When malignant disease attacks these parts it is generally by secondary diffusion, or by extension from the bones or other structures adjacent.

In paraplegia from compression of the cord by tumours, pain is, with but rare exceptions, a prominent and characteristic symptom. Some writers have expressed an opinion that it is present only when the membranes or surrounding structures are implicated, and not when the disease is strictly limited to the cord itself. This does not appear to be a well-founded distinction. In a case recorded by Mr. Shaw, in the 'Transactions of the Pathological Society' (1848–9), paraplegia was produced by two scrofulous tubercles occupying the *interior* and lower part of the spinal marrow, and *invested all round by a thin layer of medullary*

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[1] Reprinted from the 'Guy's Hospital Reports,' 1856, p. 143.

matter; yet the patient complained so much of pain in the lumbar region that it was thought her symptoms might arise from caries of the vertebræ. After death the membranes and bones were found healthy. The character of the pain appears to be very variable. In one case, quoted by Abercrombie, the first symptom was neuralgic pain in the arm, which diminished as paralysis came on. In another the patient had sciatic pain extending to the toes. Mostly the pain is referred to the back, and more or less correctly indicates the seat of the disease, from which it radiates in the direction of the nerves whose roots are invaded. Where there is no actual pain there may yet be other modifications of sensation, as coldness, or heat, or sudden alternations of these, and many other varieties of impaired feeling.

Next to pain is the frequency of muscular contractions in the affected limbs, followed as the case progresses by flexion and rigidity, and attended by a great susceptibility to the excito-motor stimulus. These phenomena are most apparent where the cord is merely stretched or compressed, and where no other change has occurred in it beyond atrophy, the communication with the brain being at the same time not entirely destroyed. If there be inflammatory softening of the substance of the cord, then these more characteristic symptoms may be absent, as they are also in compression of the cord from fracture when its structure is bruised and softened. Spasmodic contractions of the muscles of the extremities occur whether the fibres of the cord are compressed by tumours on its surface or stretched by tubercle deposited within it. At one stage of a case there may be rigid extension, which may be gradually followed as the case progresses by as rigid flexion, though the muscles at the same time may become atrophied and flaccid.

The vagueness of the early symptoms, in these as in other cases of paraplegia, deserves especial consideration in a practical point of view. In the first case here recorded, the early symptoms, cough and slight dyspnœa, and some pain in the back and shoulders, were referred to tubercular disease of the lungs. In the second, the spasmodic action of the limbs was so great that for a time the case was regarded as one of hysteria. In fine, the symptoms of neuralgia, hysteria, lumbago, rheumatism, phthisis, colic, renal calculus, pleuritic and hepatic affections, may rise like so many phantoms, to delude us at the onset of most paraplegic affections, and the errors they are apt to lead to can be avoided only by a rigid inquiry. Of the diseases of the nervous system in general, and of paraplegia in particular, it may be said that there is no symptom or single group of symptoms which, taken alone, can serve as a secure basis of diagnosis; the whole particulars included in the clinical history and present state of the patient must be viewed in their relation to each other and to *time*, before we can discern the truth they indicate.

CASE I.—PARAPLEGIA.—*Tumour growing from the inner surface of the dura mater of the cord. Early symptoms, simulating incipient phthisis; subsequently rheumatism.*

Francis H—, æt. 30, a married man of temperate habits, by trade a baker; admitted into Guy's Hospital January 30th, 1850, with symptoms supposed to be due to incipient phthisis. He had had cough and shortness of breath for two months, lost strength and flesh rapidly, and had frequent perspirations. The cough was accompanied by pain in the upper part of the back and in the right shoulder, but the complaint he made of it was not such as particularly to arrest attention. The heart's action was normal; respiration 24. He was ordered to take cod-liver oil. There was nothing specially noticed in his symptoms until the 4th of February, when he suddenly found himself unable to empty his bladder; he was relieved by the catheter, and was not for some time again troubled in that way. A week subsequently the report says, "He is improving under the use of the oil." On the 20th the pain in the right shoulder became much more severe, and he complained of feeling very languid, though up to this date he was able to walk about a good part of each day. He had occasional rigors and profuse sweatings. The increase of his symptoms at this time was attributed to his having taken cold. The next day the knees were painful, and the legs weak, he could not support himself, though he had the power of moving freely in bed. The character of the affection of the joints was such as to induce the belief that he was now labouring under rheumatism, and he was treated accordingly, apparently with good effect. At this time the inability to pass water returned, and decided symptoms of paraplegia came on, with impairment of sensation as high as a line round the chest, corresponding to the third rib; the boundary of the anæsthesia was not, however, sharp and defined. The arms were slightly enfeebled. The paralysis of motion in the lower extremities and sphincters became complete, but he retained the power of distinguishing the seat and direction of superficial impressions on the skin, though no amount of pinching or pricking gave rise to pain. The spinal column was straight, and no tender spot could be discovered on percussion, nor by the application of a hot sponge. On the 9th of March the urine was ammoniacal and contained blood. The rigors continued, and he had constant and profuse sweatings. A large slough formed over the sacrum. The pulse became frequent and feeble. He died rather suddenly on the 22nd, about four months from the date of his first symptoms.

Sectio cadaveris.—Cord softened to the extent of three quarters of an inch opposite the first dorsal vertebra. A careful examination of a transverse section showed the softening to be general, the anterior parts not being apparently more affected than the posterior. The softening seemed to be due to diminished nutrition from pressure, no traces of inflammatory exudation being detected by the microscope. The arachnoid was healthy. Attached to the inner and anterior surface of the dura mater, opposite the softened portion of the cord, was a small tumour of the size of a hazel nut. It was vascular, and consisted of nucleated cells and free nuclei in a slimy, albuminous blastema. An irregular cyst existed in the centre of the mass. The bones and ligaments of the cord were healthy. The bases of both lungs were consolidated by pneumonia of recent date; there was no trace of chronic disease. Liver soft; tissue injected. Urethra healthy. Four false passages into the bladder. The pelves of both kidneys and the bladder full of a bloody purulent secretion. The mucous membrane dark and sloughy. The secreting portion of the kidneys variegated with spots, and irregular lines of pus in the suppurating tubules.

Remarks.—An inspection of the chest movements, even in an early stage, and before the more decided symptoms of paraplegia came on, would doubtless in this case have elucidated the cause of the dyspnœa and cough. In a similar case which came under my notice, the patient was supposed to be labouring under ordinary bronchitis with dyspnœa, when I was requested to see her; but there the partially paralysed movements were at once obvious when the chest was exposed. I have to record another case, one of induration of the cord, where the early symptoms were referred to the chest, and supposed to be phthisical. The difficulty of detecting diffused miliary tubercles with the stethoscope, and the possibility of their existing without producing any perceptible dulness or marked flattening of the chest, favoured the erroneous inference of phthisis which was drawn in these cases from the cough, emaciation, and perspiration. The suspicion of such a fallacy, with a scrutiny of the thoracic movements which I have hinted at, would guard against a similar error. The cough of hysterical subjects, which is often accompanied with tenderness in the upper part of the dorsal region of the spine, may receive some elucidation from the early symptoms in this case. That the cough and dyspnœa had here a spinal origin seems evident, although doubtless, in the progress of the case, the impeded respiratory movements, and the consequent congestion of the lungs, had a great share in increasing the symptoms. The rigors and sweatings which characterised the middle period of the case were probably due to the secondary morbid changes in or about the urinary passages set up by the retention of urine and the injuries from catheterism, and not to stretching or compression of the structures of the cord by the tumour; at least it is noticeable that they were subsequent to the first catheterism, and in most cases they seem to be owing to secondary lesions. I shall not now venture to call the growth found in this case *malignant*, although at the time I examined it I had no doubt that such was its nature, and similar growths are so described by authors. I have reason to think that, before we pronounce so categorically on these productions, we must know more of the individual pathology of the membranes of the brain and cord. There was no trace of a repetition of the disease elsewhere, as commonly occurs in cancer. This fact, together with the frequency of such tumours in the dorsal region, where the effects of mechanical injuries are most felt, and the age of the patient, render it probable that it was, as its microscopical structure indicated, a simple growth due to some *local* cause of irritation. In a series of cases we may notice the gradations from nuclear and cellular towards fibroid, fibrous, and bony, in the character of these productions, such varieties being probably due to the rapidity and seat of the formation. In this case the structure was such as indicated rather rapid growth, and the history of the symptoms corresponded to it, ranging over a period of about *four months.* In a parallel case of tumour from the theca vertebralis producing paraplegia, recorded in the 'Transactions of the Pathological Society' (1847–8), the symptoms had a course of *five years*, and there the structure of the tumour was of a *firm consistence*;—*osseous* where it sprang from the dura mater, and at the other parts *fibrous*, with rough granular matter intermixed. With respect to treatment little is to be said. A better pathology and a more correct diagnosis will lead to better if not to more successful methods. At present there are no cases where treatment is more loosely tentative and empirical than in cases of paraplegia where the causes are obscure.

Case 2.—Paraplegia.—*Fibro-nuclear tumour (fibro-plastic) growing from the inner surface of the dura mater of the cord, opposite the third dorsal vertebra. Bones and ligaments healthy.* (See Plate I, fig. 1.)

Sarah A—, æt. 43, was admitted into Guy's Hospital July, 1855, under the care of my colleague, Dr. Hughes (whose kindness I have to acknowledge in allowing me to make use of this case). She was a healthy-looking woman, of a fair complexion, rather below the middle stature, employed as a domestic servant. In January she first felt pains in the shoulders, chest, and sides, aggravated at night, and at the time vaguely attributed to cold. She applied as an out-patient at a dispensary, and was repeatedly blistered between the shoulders without benefit. Her strength failing, she went into the country and kept her bed for a fortnight, hoping to obtain relief by rest. Her symptoms gradually increased in intensity, and she now began to suffer from spasmodic contractions of both lower extremities, but especially of the left. After a short time the legs were permanently drawn up to the abdomen; and, according to her description, the cramps and spasms extended to the abdominal muscles. On admission the legs were flexed, with the heels to the nates, nor could they be extended without considerable force. Left to themselves after extension, they were suddenly jerked, or more slowly drawn up into their former position. No affection of sensation. General health good. Respiratory and cardiac sounds normal. Catamenia regular. No incontinence of urine, but difficulty in voiding it; the secretion normal in appearance, and acid. Constipation. Tongue clean. Appetite good. Great distress, especially at night, from the spasmodic contractions of the legs and abdominal muscles. Her sex, her healthy aspect, the absence of any deformity of the spine, and the spasmodic character of the symptoms, led to the suspicion that there was spinal irritation of an hysterical or functional character, rather than organic disease. Three days after admission she had retention of urine. On the 1st of August menstruation returned normally. Her general health appeared unaffected. She still made great complaint of restless nights from the spasms in her legs, and of a burning pain between the shoulders, extending round to the abdomen. The sensibility of the lower extremities was unaffected. On the 6th she was cupped over the spine without relief. The legs were permanently drawn up to the nates. The urine dribbled slowly away. After a few days bedsores began to form over the sacrum, and the urine became ammoniacal and loaded with mucus and pus. On the 24th she had constant vomiting, and the tongue became dry. The spasms in the lower extremities decreased, but the great and incessant pain in the back, and the burning pain in the abdomen, prevented her getting any rest. Her strength gradually declined; very extensive sloughs formed over the sacrum and hips, exposing the bones beneath to the extent of several inches. She died extremely emaciated on the 15th of October, about nine months from the beginning of her symptoms.

Sectio cadaveris.—Bones and ligaments of the spine healthy. The theca vertebralis much distended with fluid. In the dorsal region it was translucent, and speckled with granular opacities on its posterior surface. Opposite the second and third dorsal vertebræ the cord was pushed backwards, and compressed and flattened by a smooth oval tumour growing from the inner surface of the anterior layer of the dura mater. The tumour had much the appearance, and was about the size, of a child's testicle. It had not invaded the textures, nor caused any absorption of the anterior columns. These were somewhat softened, and separated by the widening of the anterior fissure, but still everywhere continuous. Above and below the seat of compression, the cord, though small, had its natural firmness and form, and the tubules of the roots of the nerves and of the columns, examined microscopically, were normal. At the parts softened by pressure, the columns contained *granular matter* and *granule cells*, scattered amongst the tubules. Over the arachnoid, on the posterior surface of the cord, there were several scattered fibroid plates. The spinal fluid was greatly in excess, but became only very faintly opalescent by heat. The structure of the tumour was firm, and consisted mostly of cohering nuclei, generally oval, but in the firmer parts linear, with a small amount of intervening granular blastema, which in parts had become incorporated with the nuclei into an obscurely fibrous structure. In one or two parts near the surface of the tumour the texture was softer, and collections of granular matter and a cell-wall were formed around the nuclei. The whole tumour was vascular, and on compression gave out a slight quantity of clear moisture, but no opaque juice as in cancerous formations. The lungs, heart, liver, and intestines were healthy, but wasted. Kidneys not enlarged; their tunics slightly adherent; the surface granular, with a few obscure points of commencing suppuration in the tubules. The mucous membrane of the pelves and bladder injected, and covered with puriform exudation. The walls of the bladder thick. The lesion of the urinary organs was very moderate in comparison with the very extensive sloughing about the nates and trochanters. The mucous membrane of the rectum was covered with muco-purulent exudation, but otherwise healthy.

Remarks.—The prominent symptoms in this case, after the pain in the back and shoulders, were the painful cramps and spasmodic contractions in the lower extremities and abdomen. In a case of a similar tumour compressing the lower part of the cord, in a young woman whose case is quoted by Abercrombie from Gendrin, the patient suffered acute pain in both legs, and convulsive retraction of the toes, and the sensibility of the left foot was so exalted that the slightest touch produced a sense of laceration. It appears that the cord, when encroached upon by a tumour which lightly stretches or compresses it, reacts as a nerve does; if the disturbance of the structure be but moderate, there is spasm and neuralgia, passing on, with increase of the lesion, into paralysis and anæsthesia. Those who hold the theory that a motor function attaches to the anterior columns, and a sensitive function to the posterior columns, will find, to some extent, a confirmation of their views in this case, in which the lesion of the anterior columns was attended by an early and marked disturbance of the motor functions. The symptoms do not, however, seem to admit of so limited an explanation. In 1848 I took occasion, in some lectures then published, to show that we had no clinical facts which, fairly looked at, could be so interpreted; but that, in disease of the cord not implicating the trunks of the nerves or their roots, the motor function generally suffers first, whether the lesion be in the anterior, the posterior, or the lateral columns. My further experience has confirmed this statement, which has of late received elucidation from the experiments of M. Brown-Séquard. We cannot, therefore, form any diagnosis of the seat of disease in respect of the columns of the cord in cases of paraplegia from the loss of motility preponderating over the loss of sensation, since this happens as a constant phenomenon in all affections which are limited to the cord proper. The opinion which I have already expressed about the nature of these tumours, in the remarks appended to the last case, receives confirmation from the histology of this one. It was a vascular tumour, with a nuclear, fibro-nuclear, and partly cellular stroma, not invading the tissues around it, and not repeated in any other part, and hence probably of a simple nature.

CASE 3.—PARAPLEGIA.—*Fibro-plastic tumour developed under the arachnoid, on the posterior surface of the cord, opposite the seventh and eighth dorsal vertebræ.* (Plate I, fig. 2.)

William P—, æt. 41, admitted under care of Dr. Addison 25th of April, 1838. A moderately muscular man, of healthy family, and until the commencement of his present illness his own health has been remarkably good, though his habits have been intemperate. He never remembers to have injured his back, but his employment as a blacksmith subjects him to laborious, and often violent exertion. He attributes his present state to drinking cold water when heated five years ago, but however this may be, about that time he became generally weak, and had cough, attended with some expectoration and pain in the left side. His symptoms were not so urgent as to induce him to seek medical aid, and after three months' rest he returned to his work. This soon brought on pain in the back and left loin, with some difficulty in walking. For six months these were his only symptoms. He afterwards began to have a feeling of coldness in the legs, and occasional loss of sensation. The pain in the lumbar region much increased, and prevented his bending the spine in stooping. In June, 1837, in addition to an aggravation of all his former symptoms, there was partial loss of power in the left leg, the right, however, still preserving its integrity until the following Christmas, when it also became similarly affected, and both were frequently subject to spasmodic jerkings and twitchings. The sphincters became weak, and day by day, up to the time of his admission into the hospital, he noticed an increase in the paralysis, and of the involuntary contractions of the legs, any attempt at voluntary motion bringing on the spasms in an aggravated form. On admission the legs were completely paralysed, and also the rectum and bladder, and there was impairment of sensation as high as the crista ilii of either side. The loss of sensation was less in the left leg than in the right, where, excepting at the posterior part of the tibia, it was complete. The least contact of the soles of the feet with the floor caused a spasmodic tremulous agitation of the legs, which, even when not thus excited, were often thrown about by spontaneous spasms of the muscles. Nothing abnormal in the form or direction of the spinal column. Respiration hurried. Heart's force augmented. No abnormal sounds. Pulse 80. Tenderness on pressure in either hypochondriac region. A fortnight after admission pneumonia of both lungs set in, soon followed by depression, and he rapidly sank.

Sectio cadaveris.—A tall, wasted body. The theca vertebralis opposite the seventh and eighth dorsal vertebræ distended for rather more than two inches, and of a venous colour, from many tortuous vessels distributed upon it. When laid open the two arachnoid surfaces were adherent at this part, but elsewhere the membranes were healthy. On the posterior surface of the cord, and covered by the arachnoid, was a large elongated vascular tumour, slightly translucent. This growth had been developed in the pia mater. It consisted of a soft yellowish substance, very readily broken up, with numerous flattened cellular spaces interspersed through it. The medulla beneath was entire, but flattened by compression. The growth had not destroyed or invaded the membranous coverings. Under the microscope it was seen to consist in some parts, of fine, wavy, fibrous tissue, embedding elongated nuclei; in others the nuclei were round or oval, and only loosely held together by granular blastema. The right pleura was partially coated with a layer of recent fibrin; there was a similar exudation, but to a less extent, on the left pleura. The upper lobe of the left lung was solidified, and of a reddish-grey colour from recent pneumonia. On pressure the pulmonary tissue gave out a greyish puriform fluid. The lower lobe on the same side, and the right lung, more or less extensively throughout, were affected with pneumonia in an early stage. Liver large, structure granular from commencing cirrhosis. Spleen large. Kidneys large, tissue injected.

Remarks.—The almost complete identity of symptoms in this case with those of the preceding, though in one the tumour was on the posterior, and in the other on the anterior part of the cord, may be noticed as bearing upon the remarks before made on the functions of the columns.

The fatal affections of the chest, so common in paraplegia, have probably a pathological meaning of much wider extent than our present pathology seems to recognise. I allude to the influence of the spinal cord on the pulmonary plexus, and to the probable origin of pneumonia from paralysis, or a similar state of its centres and intercommunicating cords. If not in the present number of the Reports, I hope in a future one to illustrate this subject, by cases of pneumonia having particular characters and apparently caused by disease, as aneurism or tumour, invading the trunks of the pneumogastrics and the pulmonary plexus.

INFLAMMATION OF THE SPINAL MEMBRANES.

The more rare form of inflammation of the spinal membranes is where the dura mater is principally affected. This may arise from injuries to the column itself, or from cold, or phlebitis, or other causes. A remarkable instance is recorded by Mr. Simon in the 'Pathological Transactions' for 1855. A girl, æt. 18, had a fall, but soon recovered from its effects and walked home, a distance of three or four miles. After eleven days pain in the back came on, with vague symptoms of pain and tenderness over the body not altogether unlike hysteria. The movements of the trunk in bed were difficult. This was soon followed by numbness and twitching in the extremities, and after a few hours by paraplegia, complete in the legs and to a marked degree in the arms. The patient died on the fourth day from the beginning of the symptoms. Suppuration had taken place outside the dura mater throughout the whole length of the spine, and, as in the case below (Case 4), there was a burrowing of pus outwards along the course of the nerve-trunks towards the mediastinum and among the muscles. The inflammation in this case was limited to the outer surface of the dura mater, and appeared to have been set up by fracture of the body of the last cervical vertebra without displacement. The record of this case is accompanied by a still more remarkable one by Dr. Bristowe, where the suppuration was not limited to the outside of the theca vertebralis and to the formation of extensive burrowing abscesses in the course of the nerve-trunks, but the cavity of the arachnoid was also full of pus. In this instance the history was obscure, and the post-mortem examination threw no light upon the exciting cause. In the following case a similar state of things existed, set up, as clearly as could be indicated, by exposure to fatigue, wet, and cold. This may appear but a vague causation for so formidable a malady, but the evidence of other inflammatory affections confirms its truth. Almost every day's experience affords illustrations of pleurisy and pericarditis referable only to such a source, though pathology is at present at fault in unveiling the steps which lead to the results. To call them "*idiopathic*" is to satisfy ourselves with a term without meaning, and to call them "*rheumatic*" is to impose upon ourselves the fallacy of the "*ignotum per ignotum.*" I anticipate that we may hereafter be able to trace more of these acute affections to chronic diseases, the local influence of which is at present overlooked. Such an opinion is confirmed by a survey of already recorded cases. For instance, the first case which Abercrombie himself gives in illustration of "meningitis of the cord," and which he speaks of as "an example of idiopathic acute inflammation," was almost certainly set up by phlebitis of the cervical veins from chronic disease of the ear. Local phlebitis as a source of acute disease has not, except in the instances of the liver and brain, received so much attention of the profession as its importance deserves. In some cases of paraplegia associated with gonorrhœa, lately laid by me before the Medical and Chirurgical Society, this was shown to be their origin; phlebitis of the vesical and pelvic veins extending to the veins of the spine, and setting up inflammation of the membranes of the cord.

CASE 4.—PARAPLEGIA.—*Acute inflammation of the spinal membranes; softening of the substance of the cord.*

Charles H—, æt. 23, fair complexion, light hair and eyes; was quite well until Monday, April 21st, 1851. He spent the evening and part of the night of that day at Stepney fair, walking about for many hours in the wet and cold, and afterwards sleeping in his wet clothes. He affirms he did not get drunk nor receive any injury. The following day he was very unwell, with pain in the back, extending round the lower ribs, and with aching of all the limbs. The third day he was unable to leave his bed from weakness of the lower extremities, and numbness extending round the abdomen as high as a line an inch above the crista ilii. On the fourth day he began to lose power over the bladder, and the urine was afterwards drawn off by the catheter. He was admitted into the hospital on the ninth day from the commencement of his symptoms. There was then complete paralysis both of motion and sensation of the lower extremities, with paralysis of the sphincter ani and entire loss of power over the bladder. The legs were cool, and the skin mottled as from cold. He lay supine, with the legs extended. Breathing rather short and interrupted. Pulse 76. Tongue furred, white. On the tenth day (May 1st), towards evening, the skin became very hot, and the pulse rose to 132. Respiration 30. Abdomen tympanitic. No excito-motor movements could be produced in the legs, which lay extended and motionless, the muscles flaccid. Eleventh day (May 2nd): Hands cold. Pulse feeble, 132. Tongue dry and brown. Urine drawn off by the catheter; acid, sp. gr. 1024. Vomits green bilious fluid. Evacuations involuntary. Slight oppression of the brain. Respiration by the superior ribs. No abdominal movement. Twelfth day: Insensible. Respiration gasping. Pulse very rapid, and scarcely perceptible. Slight convulsive movements of the hands. Pupils active. Throughout the progress of the case, after admission, no twitchings or spasmodic movements in the legs, nor any to be excited by pinching or pricking the skin, nor by the application of heat. Died early on the thirteenth day.

Post-mortem examination (by Dr. Habershon).—Head not examined. On making an incision into the lumbar muscles pus was found upon the laminæ of the vertebræ. The spinal canal, external to the membranes, was filled with pus from the first dorsal vertebra to the third or fourth lumbar. There was a thick uniform coating of pus over the whole of the dura mater, but principally on the posterior aspect, except one patch about the first and second lumbar vertebræ. In the dorsal region pus surrounded the nerves as they left the canal. The dura mater was much thickened, and of a dull white colour, except in some parts, which were beautifully injected. At the commencement of the cauda equina, and about the lowest portion of the cord, there was a layer of pus. The vessels of the cord much distended with blood. The cord in the whole of the dorsal and lumbar region exceedingly soft, especially at the upper part (as high as the first dorsal), where it was almost diffluent. The grey matter was of a deep colour. There was no disease of the bones. Slight recent pleurisy on both sides. Pulmonary tissue healthy, with the exception of slight emphysema. Heart healthy.

On either side of the spine, where the anterior branches of the nerves pass forward, collections of pus extended along their course for a short distance. This was the case with the fifth, sixth, seventh, eighth, ninth, tenth, and eleventh dorsal nerves. These abscesses communicated with the pus contained in the spinal canal. The lumbar nerves were not thus affected. Liver and spleen healthy. Kidneys much congested, and the cellular tissue around them œdematous. The small intestines healthy. The mucous membrane of the whole of the cæcum, and five or six inches of the transverse colon, affected with acute diphtherite. The solitary glands in the other portions swollen. Bladder distended with urine.

Remarks.—The extent to which paralysis occurs, in inflammation of the spinal membranes, may obviously depend not only on the amount, and seat, and character of the exudation, but also upon the presence or absence of softening or other lesion of the cord itself. In proportion as the cord is involved in the inflammatory action will the symptoms usually considered characteristic of an affection of the membranes be less and less marked, and those of paraplegia predominate. It was so in this case. Within thirty-six hours from the commencement of the disease the patient was unable to leave his bed on account of weakness in the legs, and on admission there was complete loss of motion and sensation. It was also remarkable how entirely the functions of the brain were undisturbed throughout, contrasting, in this particular, with a large proportion of the recorded cases of acute spinal meningitis. These varieties are explained by the conditions which give rise to the disease, the nervous temperament of the patient, the degree of attendant paralysis, and the presence of actual disease in the brain itself.

The collections of pus in the course of the nerves show how the inflammatory action may be continued from the dura mater along their sheaths. There is a possibility of these purulent depôts being mistaken for the secondary abscesses of phlebitis, from which they are without difficulty distinguished by their continuity with the exudation upon the theca.

CASE 5.—*Arachnitis of the cord following an injury; paraplegia towards the end of the case.*

For many of the particulars of the following case I am indebted to my friend Dr. Wilks.

Frederick L—, æt. 22, a strong, muscular porter at a railway station, had his neck and shoulders severely squeezed between the buffers of two carriages, on the 20th of September, 1855. He was unable to work for three or four weeks, and felt much pain in the right arm, scapular region, and down the back, especially between the seventh and tenth dorsal vertebræ. The pain was increased by any sudden twist of the body, and extended to the abdomen. About a week before he came into the hospital he was again obliged to leave his work, on account of the severity of the pain along the spine. He was admitted under the care of Dr. Addison, February 6th, 1856. There was pain on pressure over the lower dorsal vertebræ, pain in the abdomen, and occasional tingling in the hands and feet. The abdomen itself was full and hard, with pain on suddenly turning the back, extending from the ribs below the umbilicus. Nothing abnormal was discoverable in the chest. The pulse was 78. Bowels regular, appetite defective. Tongue rather furred in the centre. He was treated by cupping, mercurials, and laxatives. On the 11th the pain in the back was increased. He had headache, and his nights were restless and disturbed by dreams. The shooting pain in the abdomen continued, and it was noted that the integuments were remarkably hot and dry. The pulse was 72, with a noticeable sharpness in the beat. From this date he became slightly affected by mercurial action, and was apparently improving. He left his bed for several hours in the day without inconvenience, still, however, complaining of his former symptoms and of pain through the chest. On the 28th he had general febrile symptoms, with cough and hurried breathing, and signs of pleurisy at the base of the left lung. The abdomen tense;—constipation. Pulse 112. Sleep disturbed by dreams, and by frequent spasmodic twitchings of the extremities. Complained very much of pain in the lumbar region, on each side of the vertebral column, and down the sacrum. On the 11th there was retention of urine. On the 13th slight delirium, and a marked decline of strength. He was scarcely able to move the legs, but the sensation *on pinching* was acute. He lay supine, sinking to the foot of the bed, his arms being too weak to help him to support himself. From this date he became rapidly worse, with much cerebral oppression. The urine drawn off daily by the catheter was ammoniacal, with large deposit of phosphates. The fæces escaped involuntarily. Frequent convulsive twitchings, both of the upper and lower extremities. Breathing hurried and laborious. Tongue dry and brown. Pulse 108. On the day before his death he lay nearly insensible, frequently moaning and sighing. Pulse feeble and irregular, 90. Urine, drawn off by catheter, copious. Fæces passed involuntarily. He died on the 17th, six months from the accident.

Sectio cadaveris.—The head was not examined. No injury of the vertebræ or ribs discovered. Spinal canal and external surface of the dura mater healthy. The friends would not permit an examination of the whole cord. The part removed corresponded to the lower cervical and eighth upper dorsal vertebræ. On opening the dura mater the arachnoid appeared remarkably thickened and flocculent, from effusion of lymph beneath it. The effusion was greatest on the posterior surface of the cord along the median line, but at the lower part of the cord it extended round to the anterior surface, and upwards for a short distance; the cord itself was not softened, nor, on repeated microscopical examination of the nervous substance at different sections, were any traces of exudation discovered. The dura mater had undergone no alteration, except that the inner layer was rather opalescent. One or two very small fibroid plates existed on the arachnoid. The flocculent effusion covering the cord presented under the microscope the usual appearances of inflammatory exudation on serous surfaces in the stage of organisation into permanent adhesions. Old adhesions over the surface of the upper lobes of both lungs. At the lower part of the left chest a circumscribed space, containing about a cupful of purulent fluid. Pulmonary tissue of both lungs stuffed with softish, yellow, miliary tubercles, equally diffused from apex to base. Heart and liver healthy. Kidneys large, the cortical portion studded with miliary tubercles. The splenic tissue similarly affected.

Remarks.—This case exhibits the more characteristic symptoms of pain attendant on spinal meningitis; pain in the course of the spine radiating through the trunk on any sudden twist, or other movement of the back; pain, with tingling, numbness, and twitchings, in the extremities; pain in the abdomen with hot and dry integuments, and probably, if more carefully noted, oscillations of temperature. Oliver considered pain having these characters as one of the most constant symptoms of spinal meningitis, but, like most symptoms, its presence is not invariable. In Case 7 there was no pain in the back on movement or percussion, and the patient asserted that even a blow of a sledge-hammer on the spine would not hurt him, he was so sound there. Yet the whole membranes of the cord were thickened and agglutinated by chronic inflammation. The effusion in this case was, as usual, under the close layer of arachnoid, and principally on the posterior surface of the cord, probably from gravitation. The character of the tubercular infiltration of the pulmonary tissue, and the occurrence of pleurisy with suppuration, must, as before noticed, be considered as having a probable relation to the state of the cord.

The following case, though not strictly admissible here, since at no stage was there paraplegia, is of great interest as an illustration of the apparently slight causes which may set up disease about the spine and cord. I am indebted to Dr. Wilks for the particulars of the case, and to Mr. Birkett for permission to record it.

CASE 6.—*Suppuration of the spinal membranes and formation of pelvic abscess after a blow on the back with the fist.*

Anthony P—, æt. 15, admitted into Guy's Hospital May 15th, 1856, under the care of Mr. Birkett. He was employed with his parents in a travelling show, and was in good health until three days before admission, when, playing with another lad, he received a blow on the back from his fist. He thought little of it at the time, but afterwards, the pain becoming severe, he applied for admission into the hospital. After the application of leeches he was so much relieved that he thought of going out, but the pain soon returned more severely, and fever ensued. An abscess formed on the right side of the sacrum, which was opened, and continued to discharge, the flow of pus being increased by pressure on the abdomen. He continued daily to get worse, with much irritative fever, and severe pain in the back. During the week preceding his death he was exceedingly restless, and often delirious, and complained of *pain in all parts of the body, but particularly in the extremities.* His head was generally drawn backwards, as in tetanic opisthotonos. On one or two occasions he had loss of power over the bladder and rectum, but had no other symptoms of paraplegia, and could move freely in bed. He died June 4th, twenty-two days from the receipt of the injury.

Post-mortem examination (by Dr. Wilks).—The external opening at the side of the sacrum passed into a very extensive subperitoneal abscess, occupying the fore-part of the sacrum behind the rectum, and extending over the ilia on both sides behind the psoas muscles. The bones were exposed, but not diseased. Although the abscess had discharged externally on the right side, it was most extensive on the left. It had burrowed up to the left side of the last lumbar vertebra, and through the sacro-vertebral foramen into the spinal canal. When the theca was opened, it was found to contain a quantity of greenish pus, spread over its inner surface, and over the cord itself. The dura mater, at the point indicated, was softened and destroyed, and the cauda equina was lying bathed in the pus which filled the sacral canal. The membranes of the cord were inflamed throughout their whole extent, and there was purulent effusion as high as the dorsal region. The dura mater was thickened, its inner surface had lost its smoothness and transparency, and was of a dull green colour. Pus could be pressed out from beneath the visceral arachnoid in considerable quantity. The cord itself was firm, and the microscope discovered no morbid condition. On opening the cranium, traces of acute arachnitis were found over the whole surface of the brain, greenish-coloured lymph being effused into the subarachnoid tissue, especially at the base. The inner surface of the dura mater, around the foramen magnum and on the adjacent part of the occipital fossa, was of a greenish colour, from lymph effused upon it. Pleuræ healthy. Lungs healthy. Bronchial tubes filled with tenacious mucus. Heart normal. Lumbar and bronchial glands slightly enlarged, and containing traces of tuberculous deposit. Kidneys and liver healthy. No peritonitis nor pericarditis.

CASE 7.—PARAPLEGIA.—*Chronic inflammation of the spinal membranes; œdema and softening of the body of the cord.*

Noah F—, æt. 46. Admitted into Guy's Hospital, under my care, June 22nd, 1855. A dancing-master, of rather spare frame and nervous temperament. His general health has been good. In early life he was addicted to venereal excesses, and had gonorrhœa several times. Has taken great exercise, and often walked long distances. Can give no account of any exciting cause of his present symptoms. Twenty years ago he had gout (?), and a return of it ten years ago. After the last attack he became subject to headache, dimness of sight, double vision, pinching pains in the neck, and numbness about the mouth. He was cupped, leeched, and blistered, without benefit, and was then ordered to the sea-side, where he soon recovered. A year ago, he noticed he could not give "the step" to his pupils so adroitly as he had been accustomed to do, but as he had no other symptom he took no notice of this, and continued to follow his profession as usual. Six months ago the sphincters became weak, and he began to suffer from obstinate constipation. His symptoms became rather suddenly aggravated three months ago, when he found, after sitting, he was unable to stand steadily for some minutes. He now began to have rheumatic (?) pains in the right arm, soreness in the soles of the feet, and numbness in the legs, with gradually increasing and permanent weakness in them; yet he was able, until a few days before admission, to hobble about with assistance. The symptoms of paraplegia came on with frequent and very troublesome spasmodic startings in the legs, and a peculiar sense of deadness round the lower ribs. *Present condition.*—He appears prematurely aged. The cranium is well formed. The features very intelligent, but expressive of suffering. As he lies in bed he can move the legs feebly, but has no power to stand. The sensibility is diminished below the distribution of the seventh dorsal nerve, and he has a sense of constriction around the lowest ribs, extending to the spine. The inner side of the right arm and forearm feels as if "asleep," and the fingers are weak. The *excito-motor* actions are produced by the slightest touch, or by the mere shaking of the bed; and even when quite undisturbed he is greatly troubled by what appear spontaneous startings of the legs, but which are really due to the involuntary passage of the urine, at intervals, through the urethra. The legs are more or less permanently flexed. The spine is not in any way distorted. There is no tenderness on pressure over the vertebræ, nor does the application of a hot sponge give any kind of uneasiness. He says his back feels quite strong, and if struck with a hammer there, it would not hurt him. There is obstinate constipation, and when an evacuation passes he is not aware of it. The urine is acid, and without albumen. He has either complete retention, requiring the use of the catheter, or continued dribbling. Pulse 92, feeble. Tongue moist, coated with whitish fur. Frequent profuse sweats. Emaciation. A few days after admission the urine became ammoniacal, and dribbled away continually, producing excoriation of the scrotum and a bedsore over the left trochanter. The rigidity and flexion of the legs increased, and rest was prevented by the continual spasms of the lower extremities, which were so violent on one occasion as to jerk him off the bed on to the floor. On the 16th of July he was attacked with sickness and hiccough, and became altogether so much worse that his friends removed him home. He was visited at intervals until his death, October 21st, 1855. During the three months from his leaving the hospital he gradually emaciated. The legs became permanently drawn up, the heels to the nates and the knees to the abdomen, the muscles flaccid and wasted. Any attempt to move them gave him great pain. The urine constantly dribbled away. The large intestines were emptied by enemata. Bedsores formed over the trochanters and sacrum, exposing the bones to a great extent. The feet became œdematous, and a large slough formed on the heel from pressure. The tongue became dry, the mouth aphthous. He had frequent vomiting. His intellect remained quite clear, and his mind tranquil, to the end. Though sensation was diminished in the lower extremities, he retained the power of telling which toe of either foot was touched. The slightest touch of the feet or succussion of the bed set the whole of the muscles of the lower extremities into increased contraction, and gave him great suffering. It was easy to see how an advance of disease into the cord would have greatly mitigated his miserable symptoms.

The cord only was examined post mortem. I was assisted by my friend Dr. Habershon.

The bony canal was healthy, except a very slight prominence of the intervertebral substance at the lower part of the dorsal region, which, though unimportant in this case, was worthy of note in reference to some cases of paraplegia recorded by Mr. Key. The whole of the spinal membranes, from the lower part of the cervical region, throughout the dorsal, and to a less degree in the lumbar region, were much thickened, and adherent together. The posterior layer of the dura mater in the upper part of the dorsal region, was indurated by bony plates between its laminæ. These, examined microscopically, presented the characteristic osseous lacunæ and canaliculi, but differing from the normal bone of the skeleton in the larger and more variable size of the lacunæ, and the less numerous and delicate channellings of the canaliculi. Dr. Wilks, who, as well as myself, examined them, noticed that the lacunæ had a disposition to arrange themselves in concentric rings, being formed into parcels or systems by fibrous columns running between them. The arachnoid was quite opaque and very thick. The pia mater also was much thickened. The body of the cord throughout the whole of the dorsal region was wasted and soft. The surface of the columns under the pia mater was translucent from granular exudation. Among the nervous tubules there was abundant granular exudation and granule cells. The continuity of the columns was nowhere interrupted. The lesions were due to chronic inflammation of the dura mater and more recent subacute inflammation of the other membranes, extending to the body of the cord. The bony plates of the dura mater were seated in the substance of the thickened membrane itself, and probably arose from the degeneration of new fibrous tissue. There were none of the opaque pearly plates so common on the arachnoid of the spine. This membrane was very thick, and its surfaces agglutinated by firm but recent adhesions.

Remarks.—The absence of pain and tenderness in the course of the spine was remarkable in a case where the membranes were so extensively affected; neither was there the exalted sensibility which Olivier regarded as pathognomonic of affections of the spinal membranes. The symptoms throughout corresponded, in a great degree, with those observed in cases of tumours producing pressure on the cord, with the important exception of there being no local pain in any part of the back. There was rather numbness than exalted sensibility, and yet withal, great pain towards the end of the case, when the paralysed extremities were moved. Cruveilhier has drawn attention to this symptom of paraplegia from spinal meningitis. His conclusion, though scarcely confirmed by this case, may, perhaps, be noticed here. He states that, "in paraplegia from spinal meningitis, there is—1st. Paralysis of the cutaneous nerves, gradually and successively invading the lower extremities, the trunk, and the upper extremities; at first limited to a portion of a limb, afterwards affecting the whole, and thence extending to another. 2nd. Muscular paralysis in the first period, from pain; and muscular paralysis with anæsthesia in the second period. The muscles are painful on pressure, in voluntary or involuntary contraction, or when moved mechanically. In the first period there is voluntary power to move the muscles, if an effort be made to overcome the pain; but after a short time the pain increases so that the will is powerless over the muscle. The tenderness of the limbs is not due to exaltation of the cutaneous sensibility, as the skin is insensible, but to a painful state of the muscles themselves."

The painful state of the muscles here described did not exist in this case, nor did the pain on moving the rigid and paralysed extremities appear to arise from the muscles, but from the state of the joints, or the parts about them, due to long-continued immobility. Though a degree of anæsthesia occurred in the advanced stage of the case, the symptoms were not, as Cruveilhier states, ushered in by an affection of the nerves of sensation, but, on the contrary, of the nerves of motion; and though it is obvious there may be cases where, from the inflammation affecting the posterior roots of the nerves as they arise from the cord, alteration of cutaneous sensibility may be an early symptom, yet we can scarcely understand how it should have any such necessary law of gradual and successive invasion as that here laid down, since it is obvious this must depend upon the locality and character of the effusion, and will vary with the case.

The apparently centric spasms which affected the paralysed extremities, and occasioned this patient so much distress, were really *excito-motor,* and due to the dribbling of a few drops of urine, at short intervals, along the urethra.

As to the causes which gave rise to this extensive chronic inflammation of the membranes, I learned, after the patient's death, that he had on one occasion a very severe fall upon the back, and after that, his symptoms gradually came on. He himself attributed his paralysis to the fatigue of his occupation.

Beginning with languor and partial loss of motion, with the entire absence of local symptoms in the spine, in a man having the occupation of a dancing-master, and who had exhausted his system in many ways, his symptoms were not unlikely to be attributed to mere debility, and to be met by tonics and stimulants, instead of being combated by such means as arrest inflammation. Taken at the onset, there is reason to believe much benefit would have followed a judicious and rigid treatment.

CASE 8.—*Paraplegic weakness of the upper and lower extremities ; wasting of the muscles ; mental confusion and delirium ; increased sensibility of the whole surface.*

Mrs. —, æt. 37, married; mother of four children. Previous to her present illness her health was impaired by an attack of cholera, and chronic diarrhœa, and probably also by habits of intemperance. About the end of March, 1850, she began to complain of pain in the back, wandering pains about the body, and of weakness and pain in the knees and ankles, supposed to arise from rheumatism. In a short time she was unable to walk, and the upper extremities became generally weak. The hands dropped, and hung flaccid and loose from the wrists. The muscles of the upper and lower extremities wasted, but especially of the parts most removed from the centres, as those of the thumb, the interossei of the fingers, and the muscles of the foot and calf. As the paralytic symptoms came on there was a general change in the mind. She became cunning, more fond of drink, and inconsistent and trifling in her manner, and at times delirious. When admitted into the hospital, June 5th, 1850, there was tremor and great mental confusion, but she retained so much consciousness as to be able to tell the number of fingers held up, and to put out the tongue when bidden, though she did it but imperfectly. On the 30th she lay supine, sinking to the foot of the bed. Pulse 120. Respiration 36. Skin hot. Tongue dry and brown. Muttering delirium. Sufficient consciousness to partially protrude the tongue when told to do so. Both hands dropped, and useless, yet she has power to move the arms. The legs are extended; she is unable to flex them, though she can slightly move the toes, and make the muscles of the legs contract to some extent by an effort of the will. Sensation not impaired. Complains bitterly when the legs are moved, and there is general increased susceptibility to pain over the whole surface. Speech and deglutition imperfect. Urine and fæces passed involuntarily. No deafness. Pupils rather large, but contract freely on the stimulus of light. From the above date the breathing became gradually more and more embarrassed, from the imperfect power to raise the lower ribs and to use the diaphragm. The respiration was entirely superior-thoracic, the abdomen falling in at each inspiration. There were slight twitchings of the hands, but the legs lay extended and motionless, nor could any excito-motor movement be produced by irritating the soles of the feet. She died from asphyxia, July 5th, 1850.

Sectio cadaveris.—The spinal cord was examined first. On laying open the theca the cerebro-spinal fluid appeared to be much increased in quantity, though still limpid. The membranes presented no obviously abnormal appearance. At the origin of the third, fourth, and fifth cervical nerves, the cord seemed, as the finger was lightly passed over it, rather softened, but a careful microscopical examination of several sections gave no evidence of any structural lesion. The grey matter was pale. Except increase of the subarachnoid fluid, and paleness of the grey substance of the convolutions, the brain was healthy. No increase of fluid in the ventricles. The phrenic nerve and the nerves of the brachial plexus on both sides were examined, and found healthy in their general and microscopic structure. The wasted muscles were pale and flaccid, but preserved their normal microscopic appearance. The parietes of the body, notwithstanding the great muscular atrophy, were covered with fat to the thickness of nearly an inch; the mesentery and omentum, and also the heart, loaded with fat. The right lung adherent to the parietes by old cellular adhesions, and by a deposit in these, of hard, fibrous, scirrhoid masses. The pleura pulmonalis, on the left side, was studded with numerous small tubercles, and partially adherent by tough false membranes. The cervical glands were affected by a deposit similar to that on the pleura. Liver large and pale from fatty degeneration. Kidneys healthy.

Remarks.—The origin of this case seems to have been a cachexia from previous disease and intemperance, leading to chronic cerebro-spinal meningitis and dropsy of the membranes, with atrophy of the cord and brain. The clinical history and post-mortem appearances indicate a relation to general paralysis of the insane, with this, amongst other points of difference, that the spinal centres were more affected than in that disease. The extreme wasting of the muscles of the forearms and hands, and of the legs and feet, whilst the paralysis was still incomplete, led to a suspicion of primary degeneration, either of the muscular fibre or of the nerve-trunks, but this was not confirmed by microscopical examination.

Increased membranous effusion, probably producing pressure, defective nutrition, slight softening, and paleness of the grey matter, were the only anatomical lesions to which the paralysis was attributable, and to what extent the centres of the cord had lost their functions may be inferred, not so much from the loss of voluntary power, as from the great muscular atrophy, the total extinction of the excito-motor actions, and death from paralysis of the chest.

As an illustration of paraplegic affections, the case belongs to the class which has been termed "encephalic," or "cerebro-spinal," as distinguished from the cases which have a peripheral, or a strictly spinal origin. It includes a large number of cases of paraplegia which come on after or about the middle period of life, where at first the loss of power is not so obvious as the want of management of the muscles; the memory becomes defective, the temper irritable; the pupils inactive, and often contracted.

CASE 9.—*Paraplegic rigidity of the muscles of the upper and lower extremities from limited arachnitis of the cervical portion of the cord, the affection of the upper extremities preceding that of the lower for some months.*

Mrs. L—, æt. 33, mother of one child, now fourteen years old. General health very good. Catamenia regular, up to the present time. Six months ago, having felt generally weak for a short time previous, and after a day's washing, went to bed as well as usual, but on waking the following morning her joints were painful but not swollen, and she was unable to move her arms. Under treatment by cupping and blisters between the shoulders she recovered in a fortnight, and returned to her ordinary duties, having no uncomfortable symptoms but pain in the left shoulder. After another fortnight the muscles of the arm again became rigid, the affection beginning in the shoulders and extending down the arms, so that at last she was totally unable to move them. For four months after this the legs were but little affected, and she could walk until within five weeks of her admission, the arms, however, remaining quite useless from the rigidity of the muscles. On admission, under the care of Dr. Addison, February, 1854, her symptoms were as follows:—She is quite unable to move either the arms or legs, except to a trifling degree, the left foot. The muscles of expression, of speech, and of deglutition, are unaffected. Slight anæsthesia, yet she complains of very severe pains in the knees and arms. The muscles are well nourished and very rigid. On making efforts to pass the evacuations or urine, the whole trunk and extremities become extended and more rigid. No pain in the spine at present, but formerly she had much in the neck and about the back of the head. Power over the bladder diminished, but the urine can be voided at will with some effort. Bowels constipated. Respiratory movements thoracic, heaving, and compact, not uniformly undulating. She complains much of a sense of suffocative constriction about the throat, and in speaking has no breath; nor can she cough, sneeze, or blow the nose. Speech unaffected. No affection of the nerves above the second cervical. General nutrition very good. Aspect healthy. Pulse 96. Respiration 16. Arms extended and rigid. The pain complained of in the joints depends upon the position of the limbs and the tension of the muscles, and is directly relieved, for a time, by changing their position. She has fits of shivering, which she calls hysterical; these are accompanied by increased dyspnœa. Frequent sudden spasmodic extension of legs and arms. No discoverable lesion in the bones or ligaments of the cervical portion of the spine. The pain complained of at the back of the head and neck, at the beginning of her illness, has not returned since the cupping. March 19th.—No change in the symptoms. The left pupil is smaller than the right, and the vision of the left eye is imperfect, and has been so for ten months. She again complains of an aching pain in both sides of the neck, near the occiput. Pulse 120. Respiration heaving and thoracic. The excited condition of the pulse is constant, and she is subject to frequent palpitation. April 1st.—General health remains good. Limbs rigid, especially the arms. She can sit upright in bed, firmly and without support, when placed in that position. The movements of the head on the atlas, and of the atlas on the axis, are free. Power over the sphincters diminished, but not lost. Pulse 90. Respiration 18. 13th.—Retention of urine. May 14th.—During the last few days she has had a severe pain in the head, so severe, she says, as almost to deprive her of reason. She complains bitterly of it. The sense of strangulation is very urgent. Pulse 48. Respiration 14. No delirium nor incoherence. Pupils both act on the stimulus of light; the right, as noted before, is the largest. Yesterday she was bled from the arm, without relief. On the 15th she was delirious, and died on the morning of the 16th.

Sectio cadaveris.—Subcutaneous tissue very fat. Voluntary muscles generally pale but healthy, except some of the fibres of the soleus, which had degenerated. Slight subarachnoid effusion on the surface of the brain. Moderate quantity of clear fluid in the lateral ventricles. Cerebrum, cerebellum, pons Varolii, and central parts healthy. Floor of the fourth ventricle rather opaque, the membrane closing it thickened and bulged from the accumulation of fluid in the ventricles. The membranes of the cord thickened and completely adherent together about the origin of the third cervical nerves. Above this the adhesion extended so as to implicate the origins of the second and first cervical, and on the right side also some of the lower fibres of the origin of the pneumogastric and lingual. The root of this latter nerve on the right side was embedded in a mass of opaque inflammatory exudation, and a similar mass intervened between the membranes anteriorly. The roots of the whole of the cervical nerves and of the spinal accessory were matted together by old thickening of the dura mater and arachnoid. The cavity of the arachnoid was obliterated anteriorly throughout the whole of the cervical region, and posteriorly also to a somewhat less extent. The cervical enlargement and the superior part of the dorsal portion of the cord to which the membranes were adherent was softened, and contained numerous granule cells. The whole of the local changes appeared to have resulted from inflammation. The yellow mass about the root of the lingual nerve consisted of dead exudation, and contained the débris of inflammatory corpuscles. The other smaller mass intervening between the membranes consisted in parts of cells and nuclei, and in part of fibre cells, and in one portion was organising into distinct areolar tissue. The bones and ligaments were healthy. Lungs and heart healthy, except slight and old adhesions about the centre of the left lung, and a white patch on the surface of the right ventricle. The liver contained an excess of fat. Kidneys congested.

Remarks.—There are few points in paraplegia which present more difficulty than the determination at the bedside of the causes which have given rise to the disease. In a large proportion of cases some event, which, from an accident of time only, has associated itself with the accession of the more marked symptoms, is the prominent one in the mind of the patient. It was so in this case; no account could be given of a reliable cause of the inflammation, though from the anatomical conditions of the membranes post mortem it may be inferred that some local injury set it up. It is worthy of note that cupping and blistering at once removed the acute symptoms of the first attack, and it is probable that due care would have obviated further consequences. The case was characterised by rigidity and extension; the muscles continued to be well nourished. The principal pain complained of by the patient was from the pressure of the extremities upon each other, from the tension of the muscles, which was for the time relieved by changing their position. The upper part of the cord was clearly indicated as the seat of the disease by the pain in the neck and the sense of constriction around the throat, as well as by the paralysis of the wall of the chest, the patient being unable to cough or sneeze. As frequently happens where the upper segments of the cord are affected, the arms were paralysed for some time before the legs. The irregularity of the pupils is probably referable to implication of the roots of the upper cervical nerves in the inflammatory thickening. Such a symptom is of interest in diagnosis, and one often misinterpreted as due to cerebral disorder when its source, as proved by modern physiology, may be entirely spinal. The acute affection of the membranes of the surface and of the ventricles of the brain which ended the case is worthy of note, as associated with the lesion of the membranes of the cord. It was also remarkable how suddenly, with the supervention of the cerebral symptoms, the pulse, previously ranging from 90 to 120, fell to 48.

CASE 10.—*Paraplegia coming on suddenly after fatigue and exposure to cold, and unattended by any derangement of the general health; softening of the cord in the dorsal region.*

John H—, æt. 20, a healthy, florid young man of the middle stature, occupied as a brickmaker. On the 18th of July, 1855, he walked twenty-eight miles to look for work, and slept in a brick-field. The next day he walked thirty-two miles. The day was close and wet, and he allowed his wet clothes to dry on him, without feeling any immediate inconvenience. The following morning (July 20th) he was quite well, and went out to see a cricket match. He had no stiffness in the limbs nor pain in the back. He took his dinner as usual, about midday, and in the afternoon, whilst sauntering in his garden, his legs suddenly gave way under him, and he fell down. He was, however, able to get up again without assistance, and to return into the house. About two hours afterwards he walked upstairs to his bed, feeling, as he says, all the time, "pins and needles" from the thighs to the feet. Retention of urine came on at this time, and the bowels were quite inactive. About a fortnight before his attack he had some slight warning, in not being able on one occasion to pass his water for twelve hours, but from that time until the sudden accession of his symptoms in the afternoon of the 20th he had no further inconvenience of any kind. About seven months since he had a chancre, but no secondary symptoms. Never had stricture. On admission, under the care of the surgeon (July 26th), there was complete paraplegia. Involuntary twitchings and spasms of the legs towards night. Slight excito-motor movements on touching the soles, but not on irritating the skin of other parts of the feet or legs. Gradually increasing anæsthesia below the umbilicus, but nowhere complete. Bladder much distended, with slight dribbling of urine. No priapism at the onset of the symptoms. He lies on his back, with his legs extended and the hands under the head, with an air of entire indifference as if nothing ailed him, and says he does not feel in any way ill. Tongue clean and moist. Appetite good. No headache. Pulse 90, beat sharp. Respirations 21. Motions of lower ribs imperfect. For the last two days he has had some pain in the loins, but none previously. The spine is normal. Slight tenderness about the third and fourth lumbar vertebræ. A large bulla on the sole of the left foot from hot applications. Empl. Lyttæ lumbis; Jul. Hyd. Bichl. ʒj, ex Dec. Sarzæ, ter in die. July 28th.—Complains of a sense of burning in both legs below the knees. Excito-motor action well marked in left leg, much less in right. By straining his abdominal muscles he can force a little urine out of the distended bladder. Urine contains mucus, and is alkaline from ammonia. Pulse 100. Respiration 28. Skin cool. He says he should be well if only he could move his legs. *Vespere.*—Had a slight rigor about midday. Skin hot. This rigor was probably due to a false passage made yesterday by the catheter. 29th.—Return of rigors. Legs extended and entirely paralysed, with now and then a slight involuntary jerk. The electro-contractility, even with weak currents, well marked, the electro-sensibility reduced to a perception of a faint tingling, even when the interrupted current is powerful. August 7th.—Sensation of the legs slightly returning; this is most marked in the right leg. He has no sense of tightness round the waist. Excito-motor movements are now more readily produced, and follow not only when the soles of the feet, but even when the skin of the insteps and over the legs is nipped sharply. Tongue clean. Appetite good. Skin cool. Pulse 80. Urine pale, contains mucus, and is alkaline from ammonia. 10th.—Rigors, sickness, hectic. Urine highly ammoniacal. Sloughs forming over sacrum. Rapid emaciation. 18th.—Rapid failure of strength. Frequent vomiting. Pulse 120. Skin clammy. Died exhausted, without delirium, August 20th, 1855.

Sectio cadaveris.—Body greatly emaciated. Several small superficial sloughs over the sacrum. On opening the spinal canal the sheath of the cord appeared to be more distended than usual; the inner surface of the dura mater rather opalescent. There was no abnormal adhesion nor any effusion of lymph upon the membranes; they had generally an anæmic appearance. At the middle of the dorsal region there was marked softening of the cord, with slight enlargement. The softening was most marked for the extent of half an inch about the origin of the eighth dorsal nerve, but in a less degree for an inch above and below this point. There was no apparent vascularity about the part. On a transverse section the posterior columns were quite diffluent, the anterior softened but retaining their form. The grey matter was mottled by injection of its vessels. The columns were opaque white. As a general examination of the body was not permitted, the kidneys and urinary organs were removed from behind. The kidneys were large; weight 17 ounces; the texture soft, and mottled by purulent infiltration into and amongst the tubules. Mucous membrane of the pelvis congested and ecchymosed. Bladder full of purulent and ammoniacal urine. Its lining membrane inflamed and sloughing. There were three false passages from the urethra into the bladder. One of these communicated with an extensive abscess behind the bladder, and another with a smaller abscess situate to the right side of the membranous portion. On a microscopical examination of the cord the posterior columns were found to be the seat of exudation in the form of irregular masses of granules, either free or collected around softened and broken-up nerve-tubules, and of granule-cells scattered throughout the dorsal and the lower part of the cervical region. The extent of this change was much greater than was indicated by the softening visible to the naked eye. The surface of the columns contained more exudation than the more central parts, and the white substance more than the grey. The slight mottling of the grey matter was due to injection of loops of capillary veins.

Remarks.—The striking feature in this case was the sudden occurrence of the paralysis without any local symptoms of pain or uneasiness about the spine, neither was there at any time that sensation of a band-like constriction round the abdomen which is often characteristic of disease in the dorsal portion of the cord. From this and other causes, it seems probable that this symptom is more marked when the membranes and parts about the cord are affected, than when the lesion is limited to the nervous tissue only. It may have, probably, three different sources: it may arise from a subjective state of the spinal centres at the seat of disease, referred by the patient to the course of the nerves arising there; or from distension of the abdominal viscera as a result of the paralysis; or from disturbance of the muscular action of the diaphragm, and paralysis of the lower intercostals.

The absence of all constitutional symptoms during the early part of the case was remarkable. The patient had an air of entire indifference, and insisted that he felt well but for the paralysis of the legs. If the law proposed by Duchenne had been relied on, it would have led to a grave error in diagnosis; this author having given it as a test of hysterical paralysis, that electro-contractility is unimpaired, whilst electro-sensibility is lost, yet this was the case here, on the ninth day of the symptoms, with acute softening of the cord.

If we may conclude from the presence of granular matter and "granule-cells" that the softening was the result of an inflammatory process, the amount of solid exudation is still remarkable. It seems probable that there is some prior disturbance of nutrition of the nervous tissue, of which the traces of inflammatory action are but an after result. The pathological conceptions we may form on this point are not unimportant, for at present the theory of inflammation which obtrudes itself where acute lesions of structure occur, suggests such means of treatment as not only clinical experience but the anatomical conditions themselves show to be very doubtful. Looking at the anæmia of the membranes, the œdema and softening of the columns, the small amount of exudation, without any traces of plasticity, it seems probable that a supporting rather than a depletory system of treatment is most likely to favour repair ; certainly the indications, both clincal and pathological, are opposed to the old empiricism, with its cupping, and blistering, and calomel.

Why the dorsal segments of the cord should be so frequently the seat of this form of softening is worth inquiry. It is a part where injuries are most felt, and probably the reparative power is less than in the lumbar or cervical regions, where the segments are more highly organised. Death resulted in this case from pelvic imflammation and abscess, probably not altogether independent of false passages made by the catheter.

Case ii.—Paraplegia.—*Subacute softening of the cord in the cervical region ; large osseous and fibroid plates on the visceral arachnoid.*

Bridget C—, æt. 30. Wife of a labourer; mother of four children; no miscarriages. Always had good health until six months ago (the fifth month of her last pregnancy), when she began to have pains in her knees and feebleness of gait. With these symptoms there was also some pain in the neck, between the shoulders, and down the back. The hands became slightly numb, their grasp feeble, and the muscles rapidly wasted. The legs were œdematous, and it was with great difficulty she continued to walk about until her confinement. Her labour was tedious, but accomplished naturally. The child was stillborn. Since her confinement, now five weeks ago, she has not left her bed. She has but slight power over the movements of the legs. The urine has continually dribbled from her, and there has been but imperfect control over the rectum. When admitted she was supposed to be labouring under paralysis of the bladder, from the effects of her recent labour. Two pints of highly ammoniacal urine were drawn off by the catheter. The respiration was said to be natural. Pulse 100. She was in a very helpless state from the paralytic weakness of the legs and arms. The dyspnœa and thoracic oppression were painfully urgent ; she said she felt as if she wanted the space of the whole room to breathe in. She lay supine, and preferred the horizontal position, objecting, so far as she could spare breath to do so, against being placed in a more upright position. Pulse 120. Respiration 30, with noisy bronchial wheezing. Muco-purulent secretion from the conjunctiva partially gluing the lids together. Cough feeble and ineffectual. Expectoration very difficult, muco-purulent, frothy, and viscid. Urine drawn off by the catheter, high-coloured and alkaline, with mucus and phosphates. Bedsore forming over sacrum. Intellect clear. Face livid. Feet warm. No anæsthesia either in extremities or trunk. Sense of weight and constriction over chest. December 6th.—Symptoms of bronchitis set in this morning, with great oppression of the breathing. No expectoration. 7th.—Expectoration viscid and muco-purulent. Tongue furred. Pulse 100, very feeble. 8th.—At this date the patient was placed under my care. It was now obvious from her respiration that all the intercostals were paralysed. Instead of the chest expanding in her efforts to inspire, the walls of the thorax fell in to a marked extent with each descent of the diaphragm. 12th.—Horizontal and supine position the same. No power to move in bed. Great dyspnœa. Respiration 44, entirely diaphragmatic. Expectoration very difficult, muco-purulent. Pulse 150. Skin hot and perspiring. Muscles of upper extremities much wasted, but she can lift the arms over the head. Slight anæsthesia of the fingers of both hands. No involuntary movements of the legs. Muscles flaccid. Abdomen distended. 14th.—Somewhat relieved of the dyspnœa by the use of sulphuric ether and brandy mixture. The expectoration lost for a few hours its puriform character and became serous. She died on the 18th, from gradual obstruction to the respiratory movements and accumulation in the bronchial tubes.

Sectio cadaveris.—Body moderately well nourished. Commencing bedsore over sacrum. Head not examined. Spinal canal free from disease. No inflammatory products nor abnormal adhesions of the spinal membranes. Numerous fibroid and osseous plates, some unusually large (six lines in length by four broad), on the visceral arachnoid, mostly on that of the posterior surface of the cord, and almost limited to the dorsal and lumbar regions. Many of these contained the lacunæ and canaliculi characteristic of true osseous structure. Others had partly a fibroid and partly a hyaline basis, with nuclei and lacunæ in it. The substance of the cord at the origin of the fifth and sixth cervical nerves was much softened. The softening principally affected the posterior columns and the posterior half of the left lateral column. The tissue was flocculent, and filled with granular matter and granule-cells. The disorganisation had most advanced at the surface of the cord, which was of a faint ochrey tint. The vessels of the pia mater at this part were full of blood. The principal softening was very much limited to the point indicated, but for three or four inches higher up granule-cells were found scattered amongst the fibres of the posterior columns. Lungs healthy. Bronchi full of muco-purulent secretion. Heart and pericardium healthy. Kidneys healthy, 9½ ounces avoirdupois. Spleen 4 ounces. Pelvic organs healthy. Lining of bladder apparently healthy.

Remarks.—The cause of the softening is in this case, as it is in most others, obscure. Contrary to the statement of those pathologists who have asserted that in acute softening of the cord the grey matter is most affected, the disorganisation had in this case advanced most at the surface; and although the disease was limited to the posterior columns, yet motion was principally affected. One of the chief points of clinical interest in the case was the error in diagnosis at the early part of it, when the paralytic symptoms were attributed to injury of the pelvic nerves, and the dyspnœa and bronchitis not recognised as the effects of paralysis of the chest.

Case 12.—Paraplegia.—*Softening of the cord, principally at the lumbar termination, but extending upwards throughout the whole length of the posterior columns; great congestion of the cauda equina; paralysis of right third nerve from disorganisation of the trunk near its origin.*

October 29th, 1855.—William L—, æt. 52. A tall man, with broad, well-developed frame, twice married, and the father of a large family. Had syphilis several years ago. At the commencement of his present symptoms had enlarged testes, for which he was treated with iodide of potassium. He dates his illness from four years ago, on getting wet and fatigued and allowing his wet clothes to remain on him, subsequently travelling to Exeter, and probably sleeping in a damp bed. Seven weeks afterwards he began to have pain in the loins and difficulty in passing his urine, which was high-coloured and ammoniacal. It was not until two months later that the first distinct symptoms of paraplegia showed themselves by weakness in the knees, and a sense of weariness in walking, which often obliged him to rest. He, however, continued to transact his business as a dye-wood cutter during the years 1852–3, and part of 1854, until at length he applied as an out-patient at the London Hospital. His paralytic symptoms were at this time attended with severe pain running down the right leg, supposed to be sciatica. At first he was able to walk from his house in Limehouse to the hospital, but soon the legs became too weak for this, and he was much troubled, especially at night, with spasmodic retraction of them to the abdomen. The paralysis now became complete in the right leg, and he continued to suffer from the severe neuralgic pain, commencing about the last dorsal vertebra and shooting down the leg to the sole of the foot. The left leg was occasionally the seat of the same kind of pain. Six months after becoming an out-patient of the hospital the sphincters failed him, and large bullæ formed on the soles of the feet. He now became an indoor patient for four months, without any obvious change in his symptoms, until about a week before his admission into Guy's, when one morning on waking he found himself unable to raise the right eyelid. October 29th, 1855, he was in the following condition:—Complete paralysis of the right leg. Can flex the left thigh to a slight extent. Œdema of both feet. Slight electro-contractility of the muscles of the left leg. Electro-sensibility above the knee on this side in excess. Neither electro-contractility nor sensibility in right leg. No excito-motor action in right leg, slight twitches of left. Pain at the last dorsal vertebra, extending down the legs to the soles of both feet. When the feet are roughly touched or pinched, the sensation is painful and burning. Ptosis of the right eye; paralysis also of the superior, inferior, and internal recti, with dilated pupil. Diplopia of objects to the left. Transient numbness in both hands, with slight permanent diminution of sensation in the right; no want of power in either. Tongue protruded straight. Deglutition good. He retains some power to empty the bladder; urine not albuminous, acid. Bowels inactive. No sloughs on back. No sense of constriction round the trunk. November 14th.—Paralytic symptoms unchanged. During the last four days he has complained much of headache over the forehead and vertex, and the pain down the back is more intense. To-day his manner is quick and talkative, with slight delirium. The urine dribbles into the bed. Bladder distended. He complains of chilliness, and yesterday there was a perceptible coldness of the left arm and hand. Respiration normal. Pulse 84. Diarrhœa. November 24th.—Has taken no food to-day. Lies in a dull and listless state, from which he can be only partially roused. Speech indistinct. Both pupils largely dilated, right inactive, left contracts on the stimulus of light. Urine drawn off by catheter, abundant, light amber colour, rather turbid from mucus, faintly alkaline. Pulse slow and labouring. Bowels inactive. November 28th.—Pulse 140, feeble. Respiration 40. Skin hot, bathed in profuse perspiration. Constant twitching of mouth, and lateral oscillation of the eyes. He has been in an entirely unconscious state for the last twenty-four hours. Died at 3 p.m. He rallied from his insensibility a few minutes before death.

Sectio cadaveris.—Body moderately nourished. Slight œdema of right leg below the knee. No bedsores. Only the spinal cord and brain were examined. The spinal membranes were generally very full of blood, but especially on the posterior surface of the cord, and about the lumbar enlargement and the cauda equina. The whole cord appeared to be rather small. The adhesions between the two surfaces of the arachnoid were more than usually abundant, and on the posterior surface of the lumbar medulla the two layers of arachnoid and the pia mater were matted together by fine cellular adhesions. At several points the dura mater was much thickened and vascular. The substance of the cord was generally soft, the greatest softening being at the lumbar enlargement, which was of a dull chocolate colour and infiltrated with granular cells. Many of the capillaries (veins) were irregularly dilated, and encrusted with oil-globules. The softening and infiltration extended along the posterior columns, which, examined microscopically, were found to be extensively disorganised. The focus of these changes in the cord was the lumbar enlargement and the posterior columns in the lower dorsal region, but even in the cervical segments, especially in the posterior columns, there were found a few granule cells and scattered or irregularly aggregated oil-globules, proving that the whole length of the cord was more or less implicated in the pathological changes. In the arachnoid of the lower half of the cord were many white fibroid plates and opaque granules, not unlike miliary tubercles, but smaller and less transparent. In the posterior columns, where the granule cells were most abundant, the capillary vessels were large, irregularly dilated, and encrusted with oil. The veins of the surface of the brain were distended with dark blood (death by asphyxia). There was a large excess of fluid under the arachnoid. This membrane was mottled with fatty deposits. The lateral ventricles large, containing about six drachms of clear fluid. In the centre of the right optic thalamus there was an irregular cavity, its surface lined by dilated capillary veins full of blood, and a soft flocculent tissue, containing oil-globules and granule cells; the whole of a dull ash colour, without any tinge of blood-pigment, and due to advancing ulcerative absorption of the tissue. The surrounding brain substance had a "worm-eaten" appearance, and presented all the stages of decay. At the origin of the third nerve, on the right side, the pia mater was much thickened, and infiltrated with old plastic matter, becoming fibrous and vascular, and containing in it degenerated nuclei, granule cells, and oil-globules. The trunk of the nerve was slightly enlarged and tough, and had a yellowish, semi-translucent appearance. Under the microscope it was seen to be converted into a fibrous cord, with scarcely a trace of nerve-tubule. The substance of the crus beneath was healthy. The opposite nerve was normal.

Remarks.—Dissipation, and the cachexia resulting from syphilis and its treatment, were probably the predisposing causes in this case, which needed only the vicissitudes of our climate to give rise to chronic lesion of the cord. The severe neuralgic symptoms, supposed to be ordinary sciatica, which attended the invasion of the paralysis in the right leg, was probably due to venous congestion of the nerves of the cauda equina, which was remarkable in this case. In support of such an opinion, I may mention having found in other instances varicose and enlarged venules in the trunks of neuralgic nerves. The diffused character of the lesion was indicated by the absence of any distinct horizontal line limiting the paralysis, the whole cord being in some degree implicated in the pathological process. The occurrence of ptosis, from thickening of the pia mater at the root of the third nerve, and the infiltration of the nerve-trunk with inflammatory exudation, deserve notice, as associated with the changes in the spinal membranes. The ulcerative softening and destruction of the right thalamus may explain some cases of paraplegia complicated with amaurosis. The mode of death by subarachnoid and ventricular effusion corresponded to the chronic inflammatory changes in the spinal membranes.

Case 13.—*Acute paraplegia; softening of cord; fatty degeneration of the intervertebral substance; fibrous plates on arachnoid.*

Mrs. G—, æt. 33, wife of a house-painter; mother of one child, and now ten weeks pregnant with a second. Up to the time of this illness always had good health, though apparently of rather a delicate constitution. Complexion fair. About midday on Monday, January 12th, 1855, whilst engaged in her domestic duties, she was suddenly seized with severe pain in the back, making her feel sick and faint. This lasted for half an hour, and then entirely left her. In the afternoon she went out, carrying her child, and returned home, feeling as well as usual, except being fatigued, which she attributed to having a cold. The next day, after passing a good night, she went about her household work, feeling very well until noon, when almost suddenly she became paraplegic. There was complete loss of sensation as high as the waist, as well as of voluntary movement, and entire loss of control over the sphincters. She was admitted under the care of Dr. Barlow February 23rd, 1855. Since her seizure there has been a gradual return of sensibility, and of some power over the right foot and ankle. There are several sloughs on the feet from the application of hot water, and much larger ones at the lower part of the spine, and over the hips. The face is pale, expression anxious. Pulse 120. Respiration 28, performed by the diaphragm and the five upper ribs, the lower intercostals being paralysed. The spine is straight and free from any irregularity. No tenderness at any spot. The urine withdrawn by the catheter is acid; sp. gr. 1025. On questioning her she denies having any feeling of constriction around the chest or abdomen, but complains of a slight sense of weight at the sternum. No distension of the abdomen. Liver extends two inches below the ribs. February 27th.—Her nights are restless. Continued hectic symptoms. Face now flushed. Tongue furred and dry. No headache. No delirium. Pupils rather contracted. Can move the right foot and ankle slightly. Left leg quite immoveable. Sensation perfect. Yesterday had a rigor, repeated at bedtime. March 3rd.—Sloughs on back and hips extending. No complaint of pain. Tongue dry and brown. Stiffness between the shoulders, and aching pains down the back of the arms. Occasional spasmodic twitchings in the left leg, otherwise both are motionless. *Supposed* diarrhœa due to the constant passage of semi-solid fæces through the paralysed sphincter. 7th.—Great emaciation. Pulse 130. Respiration quickened. Slight cough, imperfect from the paralysis of the lower intercostals. Muco-purulent expectoration. The passage of the urine and fæces can still be felt, but is quite involuntary. 10th.—Rapid decline of strength. Upper part of the trunk and arms perspiring. Skin of lower extremities dry and harsh. The paralysis of motion remains as before. Sensation throughout the paralysed parts nearly perfect. The smallest point can be felt, and the distance between two points appreciated as in health; yet the acuteness of the pain from pinching the skin is diminished. The symptoms were noted from day to day, but did not vary in any essential respect. She died on the 18th. The pulse was imperceptible at the wrist for many hours before death. No delirium or incoherence throughout the whole course of the disease, which lasted nine weeks and three days.

Sectio cadaveris.—Body much emaciated, with extensive sloughs, as described in the report. On removing the cord with its membranes from the canal, the parts were healthy, except a small amount of opaque, cheesy matter, oozing from the fifth intervertebral substance. The dura mater was healthy. The arachnoid was free from adhesions, and everywhere normal, with the exception of many ossific plates scattered over its visceral layer, especially about the cauda equina. The substance of the cord was much softened, from the tenth dorsal vertebræ upwards for six inches and a half. The posterior columns were diffluent, the anterior were continuous. The left column was more softened than the right. The lower section of the softened part, for about two inches, was of a dull pink colour, and the vessels of the grey matter much injected. There was no trace of effused blood. Amongst the softened tissue of this part there were a few granule cells of various sizes, and here and there an exudation cell having the ordinary appearance; but above and below this point, the columns, though soft, gave no traces of corpuscular exudation, the texture being simply loosened. The amount of exudation, even at the point of greatest softening, must have been small, as the cord was not sensibly swollen. No trace of plastic exudation on the membranes. On making a section of the bodies of the vertebræ, the intervertebral substance of the fifth, sixth, and seventh was found softened, and in part opaque, from fatty degeneration of the fibrous stroma, and the cartilage cells. The degeneration was most advanced in the fifth intervertebral substance, where the adjacent portion of the bone was becoming absorbed, and the fibrous structure of the posterior common ligament had in part yielded, and allowed some of the débris of the intervertebral substance to be squeezed into the canal, and so to injure the cord. This was the yellow matter seen on removing the cord and its membranes, and at first supposed to be strumous exudation. The lungs were free from all traces of tubercle. The lower lobes in a state of reddish-grey consolidation, easily lacerable. The bronchial membrane injected and granular, and covered with tenacious puriform mucus. Liver weighed four pounds avoirdupois; tissue pale, fatty. Kidneys soft; tissue coarse; weight ten ounces avoirdupois. Mucous membrane of bladder inflamed. Sloughs extending through the anterior wall to the sheath of the rectus, and posteriorly destroying the vagina. The os uteri sloughing, and a small ovum, well formed, protruding. The mucous membrane of the small and large intestines much congested. In the stomach, at the larger curvature near the fundus, were several ulcers of the size of a sixpence, the black sloughs of the mucous membrane being still adherent.

Remarks.—The exciting cause of the acute softening of the cord in this case was mechanical injury, resulting from the giving way of the posterior common ligament, and the escape of the débris of the degenerated intervertebral substance into the canal. The sudden pain in the back, with sickness and faintness, felt by the patient on the day previous to the paralysis, probably depended upon this rupture. It was thought, when the canal was first opened, that the lesion of the intervertebral substance was due to scrofulous deposit; but that opinion was not supported by a further examination. It appeared to be only a form of atrophy, leading to opacity and fatty degeneration of the texture. As it was but a small spot of the posterior surface of the intervertebral substance which was affected, there was no displacement of the bones. The recovery of sensation even whilst the case was progressing to a fatal termination is of interest, as bearing upon prognosis. The same is observed in hemiplegia, from softening or effusion of blood into the corpus striatum or thalamus opticus, the loss of sensation accompanying the injury to the nervous centres being after a few days recovered, though in other respects the symptoms may have undergone no favorable change. The subject has also a further interest in reference to the physiology of the sensitive functions of the cord, especially, as it will be observed, that the posterior columns were broken down, and only the anterior continuous. The cause of death was exhaustion from the unusually extensive sloughing of the pelvic viscera, and of the skin over the sacrum.

The amount of exudation into the cord was, as usual, very small, and except at the part principally affected, the tissue was simply loosened and œdematous. Some of the granular bodies seen under the microscope were formed by the aggregation of granular matter around broken nerve-tubules, others had the more common origin from degenerated exudation corpuscles. Whether such a lesion as this, apart from the chronic disease of the surrounding structures, is remediable, is very doubtful; but, as observed in the preceding case, we should expect less from the use of calomel than from those means which favour nutrition.

CASE 14.—*Paraplegia preceded by symptoms of colic; sudden loss of power and sensation in the upper extremities; partial recovery for some months; relapse; general and slight softening of the whole cord; traces of inflammatory exudation discovered in the cervical portion, and in the medulla oblongata.*

Many of the particulars of this case were collected for me by my friend Mr. Edmund Galton.

Esther J—, æt. 32, a stout, leucophlegmatic woman, a widow, never had robust health, and as a girl was subject to severe headaches, and at times to hysteria, and also to painful and irregular menstruation and palpitation. Had, according to her account, two attacks of pleurisy eight years ago, and soon after was in St. Thomas's Hospital for rheumatism. Eighteen months ago had symptoms of colic, attended with giddiness and slight mental confusion; and about that time, on waking one morning, found she had lost to a great extent the power of motion and sensation in both upper extremities. The hearing became at the same time dull, and her memory impaired. She gradually recovered the use of the arms, and continued in her usual health (though not able to walk up and down stairs, and occasionally having difficulty in breathing) until two months ago, when she began to have pain in the back between the shoulders, increased difficulty of breathing, pains in the limbs, with formication in the fingers, and pain in the left side and abdomen. A fortnight after this aggravation of her symptoms the power of the upper extremities became again much impaired, the wrists dropped, and the hands became numb. The legs were less affected; she was able to stand, but not to rise from her seat without assistance. The loss of power was rather more marked on the left side than on the right. The sense of taste was lost. On admission into the hospital, August 22nd, 1855, the following note was made of her condition. Sensation in the left arm perfect as far as the elbow, below it is gradually lessened, and entirely lost in the fingers. The motion of the shoulder-joint unimpaired, but attended with pain in the back. Power of extending the elbow-joint very imperfect, wrists dropped. Right arm similarly but less affected. Can move the legs in any direction in bed, but is not able to stand without support. No affection of sensation. Loss of taste. Complains of pain in the back, passing over the shoulders. Tenderness on pressure over the lower part of the cervical, and upper part of the dorsal region. No abnormal condition of the spine discoverable. Severe griping pains in abdomen. Obstinate constipation. Dyspnœa, cough, and constriction across the chest, with inability to expectorate. Slight bronchial râles, respiratory sounds otherwise normal. Power to empty the bladder remains. Urine acid, high-coloured. Frequent cold perspirations, followed by flushing heats. Sleeplessness, despondency, globus, and other hysterical symptoms. There was no marked change until September 9th; she was very desponding, and often expressed a wish to die. Had day by day various nervous symptoms of an hysterical character. Bowels obstinately constipated, great pain in the abdomen. Slight traces of blue line on the gums, which, with her other symptoms and the dropping of the wrists, favoured an opinion of lead poisoning. The dyspnœa, cough, and inability to expectorate, with a sense of suffocation, continued to distress her very much. On the 9th all the paralytic symptoms were in a few hours increased, with intense pain between the shoulders and across the chest. The urine was passed naturally. Pulse weak and frequent. On the 11th she was universally paralysed. There was frequent cough with inability to expectorate, and an increased sense of suffocation. Cardiac sounds very feeble. Paralysis of the respiratory muscles gradually increased.

The larynx soon became involved. She was neither able to speak nor swallow, but remained perfectly sensible until her death at midday.

Sectio cadaveris.—Body well developed and stout. No sloughs nor abrasions on the back. Integuments and internal organs generally congested from the mode of death. Cerebrum, cerebellum, crura cerebri, and pons Varolii healthy. Medulla oblongata softened. Under the microscope there was seen, here and there, an exudation cell amongst the loosened tubular structure. The spine and its membranes were healthy. The cord softer than usual throughout; but it was only after examining many parts, that any trace of exudation was discovered, and that only in the cervical region. Without repeated examination this would have been overlooked, both in the medulla and in the cord. The viscera, including the kidneys and bladder, healthy, with the exception of recent congestion.

Remarks.—The morbid anatomy of this case is of great interest as elucidating those recorded instances of paraplegia where no lesion of the cord was observed. It was only by great patience that the microscope discovered any traces of inflammatory exudation, but these, though slight in amount, were distinct and decisive. It is probable the paralysis was rather due to the arrest or perversion of the normal processes of nutrition than to the mere mechanical effects of the exudation.

The early symptoms were vague, and thought by some to be hysterical, thus affording another proof that it is not in the symptoms themselves, taken individually, but in their course and grouping, that the true basis of the diagnosis lies.

Whether the paralysis was the effect of lead, as was supposed from the traces of a blue line on the gums, the dropping of the wrists, and the colic and constipation, is doubtful, as the anæmic and icterode tinge of the surface and conjunctivæ, so characteristic of lead poisoning, was wanting; neither do the affections from lead take such a course to a fatal termination. The remarks on the obscurity of causation apply to this, as to most cases of softening.

Case 15.—*Paraplegia commencing by paralysis of the right arm, and referred to an injury of the hand; an undefined nuclear growth in the cervical region of the cord, and a similar degeneration of the grey matter throughout.*

Abraham C—, æt. 23, stoker on board a steamboat, of intemperate habits, but has had pretty good health. Has occasionally been in pugilistic encounters, and received many blows on the head and forehead, but the most severe was about five years ago, when he was struck unexpectedly by another man's fist on the side of the neck, near the articulation of the skull with the vertebral column. Since that, he has occasionally had difficulty in deglutition, particularly of fluids, which would be expelled through the nose. For the last year he has had a choking sensation, and, at times, difficulty in passing water. He attributes the weakness of his right arm to a blow which he received on the back of the hand, eighteen months ago, by the falling of a piece of iron. This accident kept him from his work for six weeks, but the wound healed without any extension of inflammation up the arm. As he recovered, he noticed a want of power in the ring and little fingers, and the whole arm, from the shoulder, became wasted and weak. He continued to work with his left arm for three months longer; but about the beginning of the year 1850 he began to suffer from what he terms "bile," that is frequent vomiting, unattended by any pain in the head or giddiness. These returns of vomiting continued for four months, and then, as they subsided, there was increased difficulty of deglutition, and both legs became weak, the left first and to the greatest degree. In the autumn he improved, and was able to walk about, but the bladder was so far paralysed, that he needed the catheter to be passed for several weeks. The improvement was only of short duration. On his admission into Guy's, June 5th, 1851, under the care of my colleague, Dr. Barlow, the right arm was completely paralysed at the shoulder-joint, and there was great wasting of the muscles, only slight power of moving the fingers remained. There was anæsthesia increasing towards the hand, most marked in the branches of the ulnar nerve. No actual paralysis of the left arm, but the muscles flaccid and weak. He had pains running over the back of the head. He could move the legs slightly. Sensation impaired as high as the hips. No deformity of the spine, nor tenderness on percussion. No sense of constriction at any part of the trunk. Vision somewhat impaired. Urine and fæces often passed involuntarily. Pulse 90. Tongue clean and pale. He improved, by rest and by the use of electricity, so far that in October, he could support himself and walk without help, though the gait was very vacillating, from want of power to direct the muscles. No numbness remained in the legs. The right arm continued in the same state as on admission. The left was weak, and at times he had cramp in the muscles, and involuntary closure of the hand. The sphincters partially paralysed. Aspect pale and emaciated; the whole muscular system much atrophied. He continued in the hospital until June, 1852, his symptoms fluctuating between improvement and relapse. He could walk about the ward, by the aid of a stick, with a feeble gait, his right arm hanging loosely, supported only by the ligaments of the shoulder-joint. On leaving the hospital he went to Dover, but returned, and was readmitted in October, 1852. In a few weeks the left arm was quite paralysed, and he lost the little remaining power over the legs and sphincters, and became universally paraplegic. He often complained of a sharp pain in the back of the head and in the upper part of the neck. On 19th January, 1853, bronchitis came on from exposure in moving him from one ward to another; though trifling in amount, the distress occasioned by it was inexpressible, from the paralysis of the intercostals. A remission of his chest symptoms occurred until March 14th, when they again became aggravated. His distress was indescribable. Constant ineffectual efforts to expectorate; pulse rapid, 120; respiration 36; face congested; complete paralysis of the extremities and walls of the chest, and general anæsthesia, yet great pain when the body or limbs were roughly handled; frequent spasms in the legs; arms not so affected. Urine constantly dribbling. Slight abrasion of the skin over the sacrum, but no sloughs occurred throughout his illness. His miserable existence was drawn out until April 12th, 1853.

Sectio cadaveris.—Remarkable atrophy of the whole muscular system, and of the tissues generally. Slight abrasion of the skin over the prominent part of the sacrum. No slough. Diffused tubercular masses and scattered tubercles through the upper lobes of both lungs. Dilatation of the bronchial tubes; their lining deeply injected. Contents purulent. Heart healthy. Hepatic tissue congested and fatty. Kidneys healthy. Pia mater and brain tissue rather watery. On removing the arches of the vertebræ the whole cord appeared to be large and swollen; in the cervical region the theca was evidently distended by it. There was no affection of the bones or ligaments. On laying open the theca there was a general enlargement of the cervical portion of the cord, which, on transverse section, had an unusual appearance. The columns had a yellowish tint, and were distended by a soft vascular translucent growth, parts of which were firmer and opaque yellow (dead?). This growth was not defined, but passed insensibly into the degenerated grey matter (Plate I, figs. 3—6), which from the floor of the fourth ventricle to the filum terminale was pale and swollen, and had much the physical character and consistence of thick boiled starch. This soft starch-like substance under the microscope was seen to consist of round, oval, and elongated granular nuclei, embedded in a slimy blastema. At the filum terminale, where the more normal characters of the grey matter were preserved, these nuclei were scattered amongst the softened tubercles with exudation cells. The vascular growth in the cervical region consisted of degenerated nervous tissue, nuclei, and nucleated cells, as in the fibro-plastic growths. The opaque part was little else than granular matter and oil-globules. There was no lesion of the membranes of the cord, nor was the continuity of the columns destroyed, though in the cervical region they were spread out and slightly softened in parts. The nerves arising from the cord in the cervical and lumbar regions, examined microscopically, had the normal structure.

Remarks.—The limitation of the paralysis at its commencement to the right arm, and the preponderating affection of the muscles of the shoulder-joint, are points in the history of this case of great interest. Taken together with the injury to the hand, to which the patient attributed his symptoms, they led to an opinion that the case was one of peripheral paralysis, but such an inference was not supported by the history of the case, nor by the post-mortem appearances of the cord. The slight affection of the muscles of deglutition, the sense of choking, and the occasional loss of power over the bladder, connected the blow at the upper part of the spine with the lesion in the cord, whilst the peripheral origin of the malady was entirely negatived by the normal microscopic structure of the nerve-trunks. The general atrophy of the muscles of the extremities in the progress of the case before the more distinct symptoms of paralysis occurred is deserving of special note, as bearing upon the theory of progressive muscular atrophy, many examples of which have no doubt had, contrary to the opinion of those who recorded them, a spinal rather than a muscular origin. The atrophy of the muscles of the right shoulder, whilst those of the forearm still retained some power, elucidates the seat of the paralysing lesion in some cases of infantile paralysis of the shoulder occurring during dentition. It has been doubted whether the lesions alluded to have a cerebral or a spinal origin, but their occurrence without any cerebral symptoms, the occasional affection of both arms or of all the extremities, and the actual observation of a limited spot of ochrey discoloration in the cord, as in one case examined by Cruveilhier, concur with the collateral evidence here afforded in proving a spinal origin of this form of paralysis. There is nothing in practical medicine more fallacious than hastily inferring a negative from negative evidence, as was proved in this case. The absence of pain on percussing the spine, and the positive account given by the patient that his paralytic symptoms were the result of the injury to the hand, led to the conclusion that no lesion of an active kind was going on in the cord, yet we can have no doubt that the contrary was the fact. The least consideration will show that if the ligaments and bones be healthy, no amount of pressure or percussion made in the usual way of a clinical examination can much affect the structure of the cord itself, and that we can base no inference upon the negative evidence so afforded. There is a minor symptom in this case deserving of notice; I allude to the impairment of vision. This may be associated from different causes with paraplegia. Here it was probably referable to the changes in the cervical portion of the cord itself, since it has been clearly shown by experiments on animals that the condition of the eye is at once affected by injuries to the roots of the cervical nerves. I have had occasion to notice this in preceding cases.

Vomiting, as an early symptom of disease of the cervical portion of the cord, occurred in this case, and was probably dependent upon the origin and connection of the phrenic nerves; in another case, as we have seen, the symptoms set in with an irritating cough. I remember an obstinate case of hiccup, which, having resisted other treatment, yielded at once to blisters on either side of the cervical portion of the spine, over the origin of the phrenic nerves. The character of the local changes in the grey matter was peculiar, and probably depended partly upon degeneration of the normal structure, and partly on a neoplastic formation of the simplest kind. In the cervical region, where the disease first commenced, this had progressed to the greatest extent, making an approach to the development of a tumour, but not separated by any line of demarcation from the other parts of the grey matter which had undergone a similar, but less advanced, change. The existence of tubercles in the pulmonary tissue can hardly, in cases like this, where young persons have been long bedridden, be regarded as an index of a previous scrofulous habit, since it is more than probable, at least in some of the cases, that the tubercular diathesis was induced by the unfavorable circumstances to which they were subjected.

CASE 16.—*Paraplegia; early symptoms referred to rheumatism and phthisis; induration of the cord at the cervical enlargement; softening of the dorsal segments adjacent.*

For the following case I am indebted to my friend Mr. Bradley, who also kindly sent me the cord for examination. The patient was an inmate of the Model Prison.

W. P—, æt. 29, a single man, native of Devon, employed as a shepherd, of healthy appearance and florid complexion. His health had always been good previous to the 25th of January, 1850, when he began to complain of pains which he attributed to having caught cold in a bath some days before. These pains at first occupied the left shoulder, particularly the scapula and the deltoid, but subsequently extended down the arm to the fingers, and at times wandered into the leg of the same side. From the description given of the pains, and from the absence of other symptoms, either local or general, the case was regarded as rheumatalgia. During a period of two months various remedies were in turn prescribed, but without any satisfactory result. The only one that afforded any relief was morphia. By this time the pain was localised in the shoulder-blade, and though not paroxysmal, yet, from its severity, it appeared to be neuralgic. Symptoms of phthisis also now began to show themselves. There was great emaciation and muscular debility. Pulse 120 and weak. Profuse nocturnal sweats, especially about the head and chest. Dry cough. Chest everywhere resonant; respiratory murmur at the left apex impaired. Constipation. By the 7th of April the weakness of the lower extremities had increased, so that he was unable to stand. There was imperfect control over the sphincter ani, and the urine was retained. That drawn off by the catheter was clear and faintly alkaline. On the 8th the urine was ammoniacal. Motions passed unconsciously. Voluntary movement of lower extremities lost. Sensation impaired from fourth rib downwards. Upper extremities unaffected. Intellect unimpaired. No tenderness on percussing the spinal column. Appetite unimpaired. Sleep sound. No complaint of pain. The treatment consisted in the exhibition of calomel in grain doses to affect the system. Blisters, with mercurial dressing, to the lower portion of the cervical region of the spine, and between the scapulæ. On the 18th his condition was much the same as on the 8th, except that sensation in the lower extremities was improved. There were cramps and flying pains in the limbs, with spasmodic movements of the muscles of the upper extremities. The perspirations were profuse. Gums affected by mercury. May 1st.—A seton was inserted on both sides of the spine, at the nucha. On the 13th the cough and expectoration had ceased. Sensation had returned in the legs; he could retain his motions, and was conscious of the passage of .the urine. On the 23rd the setons were removed, and, as he complained that the involuntary spasms of the limbs prevented sleep, he was ordered half a grain of morphia every night. On June 3rd he had lost power in his hands, and there was loss of sensation in the ulnar side of the right hand and back of the arm. The paralysis of the lower extremities continued, but sensation was restored, and he could retain the urine and fæces. Subsequently the pain and spasms of the lower extremities were very distressing, and he complained of pain about the third and fourth dorsal vêrtebræ, increased by percussion. On the 20th bronchitis set in, attended with great difficulty in expectoration. He gradually sank on the 29th.

Sectio cadaveris.—Body greatly emaciated. Brain and its membranes healthy. Membranes of the cord healthy. At the cervical enlargement the cord was indurated to the extent of an inch. On section at this point the columns had a greenish-yellow tint, and were of an almost horny hardness; below this part, for three or four inches, there was marked softening, the columns being nearly diffluent. Kidneys large, pelves and ureters dilated. Abdominal viscera healthy. Recent pleuro-pneumonia at the bases of both lungs. The apices adherent, the pulmonary tissue indurated, and containing several small vomicæ (?). Bronchial tubes generally dilated, and containing muco-purulent secretion.

Remarks.—I have to regret that a microscopical examination was not made of the indurated portion of the cord in this case. The induration was uniform, and the cord somewhat swollen, as if from fibrinous infiltration of its textures. The chief clinical interest of the case was the obscurity of its early symptoms. For a period of two months pains, supposed to be rheumatic or neuralgia, were the only complaint, and it was even suspected that the patient, under the circumstances, might be feigning illness. The next phase was great muscular debility, rapid emaciation, dry cough, and profuse sweats. As the chest was resonant throughout, the patient was supposed to be labouring under diffused tubercular disease of the lungs, though there can be no doubt the symptoms had, as in a previous case (Case 1), a spinal origin. It was not until imperfect control over the sphincters roused attention that the spinal disease was suspected. It will be observed that after the first shock of the onset of the more marked paralytic symptoms sensation slowly returned, as in a previous case. The spinal membranes were healthy, yet it will be observed that the early symptoms were pain, radiating in the course of the nerve-trunks, and as the paraplegia became more marked the patient was greatly distressed by painful spasmodic contractions of the legs. There was no account in the history of the case of the exciting cause of the malady.

PLATE I.

Fig. 1.—A vascular fibro-nuclear tumour growing from the inner surface of the dura mater on the anterior part of the cord in the upper part of the dorsal region: the cord pushed backwards and compressed (Case 2, p. 170).

Fig. 2.—A vascular fibro-plastic tumour, situate on the posterior surface of the cord in the lower part of the dorsal region under the close arachnoid (Case 3, p. 172).

Figs. 3—6.—Sections of the cord in Case 15, p. 199, showing the enlargement (6) in the cervical region from the development of a soft vascular nuclear growth in the grey matter. The sections 5, 4, 3, are intended to show the same in a less degree in the other parts of the cord lower down.

Plate 1.

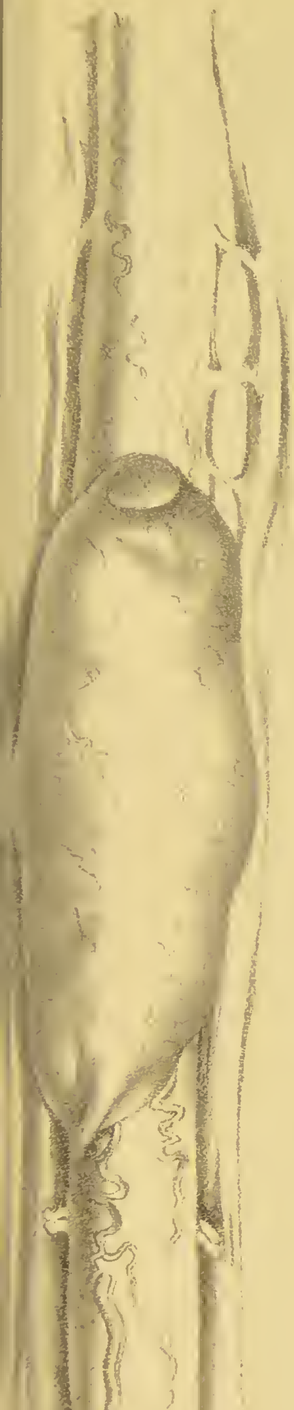

Fig 2.

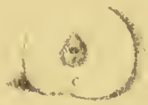

Fig 3.

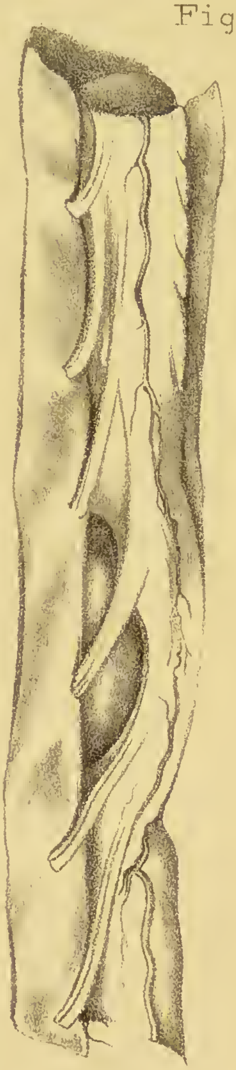

Fig 1.

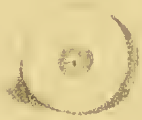

Fig 4.

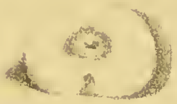

Fig 5.

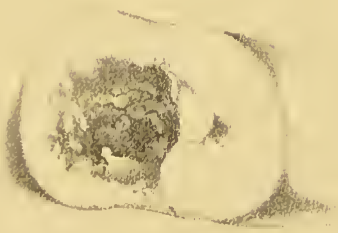

Fig 6.

West, Newman lith.

CASES OF PARAPLEGIA.[1]

(SECOND SERIES.)

THE following cases of paraplegia, with those in the 'Reports' for 1856, though a miscellaneous contribution, may perhaps serve for reference in the absence of a more systematic treatise on the subject.

The labours of Lockhart Clarke and Lenhossék, on the minute structure of the nervous centres in health, cannot fail to give a new impulse to a more exact knowledge of the pathological changes to which they are subject. Something in this direction has been attempted. Those who are acquainted with the results of minute anatomy, as applied to the cord, will admit that we may now hope for an exhaustive morbid anatomy of it, exhaustive, at least, so far as to enable us to determine the state of the ultimate tissue.

Case 17 goes far to establish an important point in the pathology of paraplegia, namely, that the spinal centres may be paralysed without anatomical change of their structure. If this were certain it could not fail to give a new direction to our inquiries, and lead us with more earnestness to investigate the nervous substance by other means than the microscope. Dr. Sankey's observation on the variable specific gravity of the brain, lets in some light in this direction. It is from an increased knowledge of "atomical," as distinguished from "anatomical" conditions, that we may hope for future advances in nervous pathology.

Case 18 presents a not uncommon history of chronic inflammatory degeneration of the columns of the cord almost latent up to a certain point, and then accompanied by a sudden aggravation of the symptoms. It may offer an occasion to remark that in diseases of the nerve-substance, acuteness of effects is no evidence of acuteness of the lesion producing them. In the brain this is notoriously true, for every one knows that a sudden hemiplegia may result from local changes of the slowest and most passive kind. The same occasionally occurs in the cord. The bearing of this on diagnosis and treatment is obvious.

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[1] Reprinted from the 'Guy's Hospital Reports,' 1858, p. 169.

Case 19 is a remarkable instance of the limitation of disease to the posterior columns. The lesion was of the same character as in the preceding case. The symptoms confirm the theory of Dr. Todd that the posterior columns are the channels through which the voluntary movements are co-ordinated. In this case there was not paralysis, but a want of controlling power. There was only a slight affection of sensation, proving also that the posterior columns are not mainly subservient to the sensory function.

Case 20 presented at the bedside a rare symptom in paraplegia, namely, paralysis of both seventh nerves. This prevented the pronunciation of the labial parts of speech, and led to a suspicion of brain disease until the kind of defect was pointed out. This accident was explained by the condition of the medulla oblongata.

As it has been just remarked, on the one hand, that the character of a lesion of the nervous substance is not to be inferred from the acuteness of the symptoms in respect to their development in time, so it may be added, on the other, that the amount of the lesion is not necessarily in proportion to the gravity of the symptoms. A very small amount of anatomical disease, or, as we seem to have proved in Case 17, not enough to be recognised, may produce fatal effects. The degree of positive lesion was appreciable in this case, but it was in amount trifling. It was its seat which gave it its importance. It is in the treatment that we need to bear these truths in mind, as no doubt there is a proneness in the mind, as before said, to estimate the activity and violence of a disease by the rapid development and danger of the symptoms, and, consequently, to aggravate it by too heroical interference.

In Case 21 the limitation of the disease at its onset to the right side of the cord, and the suddenness of the early symptoms are the chief points of pathological interest.

The occurrence of erysipelatous inflammation from the incautious application of heat, is an accident to which paraplegic patients are notoriously exposed.

It does not seem unimportant to draw attention to the difference between capillaries mechanically incrusted with fat-globules as the result of disease of the tissue in which they lie, and that form of fatty degeneration which is precursory of atrophy.

Case 22 shows that the substance of the cord may be damaged by a violent exertion, without any affection of the bones, ligaments, or membranes of the spine. Whilst such injuries have an immediate interest to the surgeon they have not less a deferred interest to the physician, who is often called upon to treat the subsequent effects. It is on this account that I have recorded this case and Cases 23 and 24. In Case 23 there was bruising and ecchymosis of the posterior columns and of the grey matter, followed by hyperæsthesia of the parts below. In Case 24 there was first paraplegia from concussion of the cord, recovery after a few hours, and subsequently fatal paraplegia from extravasation of blood outside the theca vertebralis along the spinal canal.

Cases 25 and 26 are instances of progressive muscular atrophy from chronic disease of the cord. The early symptoms of such cases are like those which come on in lead-poisoning. The wrists drop and the hands become weak. It seems hardly necessary to assert that such symptoms are not pathognomonic of the presence of lead, as some have stated, for it must be obvious to anyone who will consider the matter that in chronic affections of the cord in the cervical region the disease is not always so uniform in its seat and extension as to affect the muscles in the same order. In one case the arms may waste generally and equally throughout; in another, the scapular muscles and those of the shoulder may be first affected; and in a third, the interossei of the fingers, the short muscles of the thumb, and the extensors of the wrists may first fail. The relation of the nerves of the brachial plexus to the cervical enlargement of the cord, partly explains these differences, and what remains obscure seems to require for its elucidation only a more accurate investigation of the distribution of the lesion through the cord in particular instances.

Cases 27 and 28 are recorded for the purpose of pointing out the occurrence of acute rheumatic symptoms after spinal lesions. Mere pain in the joints and limbs, generally, is not what is here meant. It is too common an error to account for obscure pains by calling them "rheumatic," to need any remark. The symptoms referred to are commonly regarded as pathognomonic of a rheumatic state, namely, swelling and redness of the joints; profuse acid sweats; high-coloured, scanty urine depositing urates, &c. Our ignorance of the essential nature of acute rheumatism prevents our asserting or denying that it may have its origin in a disturbance of the nervous force, but certainly a condition apt to be confounded with it does so arise. In practice it cannot be an indifferent matter, whether, according to popular pathology, we set before us, as the object of our treatment, the elimination of a materies morbi or a lesion of the nervous centres.

Case 29 is remarkable for its clinical history, and the apparent contradictions which misled the diagnosis for the first two or three years. It was a case of chronic thickening of the spinal membranes implicating and destroying the posterior roots of the nerves of the brachial plexus. The disease was for some time so limited as to produce no other symptom than numbness of the left arm.

The test by galvanism, proposed by Duchenne, was entirely fallacious. This excellent author, in a *résumé* of his deductions on what he terms "faradisation" (electro-magnetism), applied to pathology, gives the following conclusions in respect to hysteric paralysis:[1]—1. Electro-muscular contractility is normal in hysterical paralysis. 2. Electro-muscular sensibility is, on the contrary, generally diminished or altogether absent. 3. Lastly, voluntary movements may be intact notwithstanding the diminution or loss of the electro-muscular sensibility.

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[1] 'De l'Electrisation localisée,' p. 530.

All these conditions concurred in this case, and yet it was one of organic lesion of a serious kind.

The case affords a striking proof of the insidious origin and course of chronic spinal meningitis. The local action appears never to have been acute, and was unattended by those symptoms of irritation which are supposed to characterise inflammation of the membranes of the cord.

The changes in the cord were probably subsequent to those in the membranes and posterior roots of the nerves.

Cases 30 and 31 are good examples of malignant disease about the spine affecting the cord. In one the substance of the cord was sloughing; in the other the proximity of the cancerous growth had induced only softening. In neither was the nervous substance the seat of the new growth. At an early period, when there is nothing tangible, the symptoms in such cases are commonly referred to neuralgia; the word "neuralgic," for explaining symptoms, as the word "idiopathic," for explaining causes, being of so easy use that it invites careless investigation. But, for this, there are generally circumstances which would suffice for a sound diagnosis. The pain is more or less characteristic in its continuance and severity. Its seat about the spine is also a sufficient cause of suspicion, since this region is not commonly affected with pure neuralgia; added to which, collateral symptoms, if sought for, are often found to remove the difficulty. For instance, signs of pressure on the bronchi, where the dorsal region is the seat of the disease, or, as in Case 30, the invasion of an adjacent organ by the malignant growth.

Case 31 is one of strumous tubercle developed towards the centre of the cord. The chief value of the case lies in its history, for the patient being an infant, as the arms only were at first affected, the paralysis might not have been regarded as of serious importance. Young infants are occasionally the subjects of paralysis of one or both arms, from the carelessness of nurses in tying the dress so as to produce pressure on the axillary plexus. Not unfrequently also the paralysis of the period of dentition, the "paralysie essentielle" of French authors, shows itself in one or both arms, whilst the legs remain unaffected. In both these forms the onset of the paralysis is sudden, and by that alone they would be distinguishable from such a case as the above.

Case 17.—*Complete paraplegia without loss of sensation; onset of symptoms sudden. Death after fourteen days from acute peritonitis set up by inflammation of the bladder; no discoverable change in the structure of the cord beyond slight softening of the texture; no exudation.*

(Reported by Mr. Durham.)

Henry P—, æt. 32, clerk to a solicitor in the City, was admitted under my care into Guy's Hospital, 23rd December, 1857. A tall, well-made, rather pallid, but otherwise healthy-looking man, suffering from entire paraplegia of the lower extremities and sphincters, but without affection of sensation. He stated that he had never previously had any serious illness, but that two years ago he fell whilst attempting to jump over some chairs. After a few days all apparent effects of this accident passed away, and he considered himself in unimpaired health. In the summer of 1857 he married, and gave himself to excessive indulgence in sexual intercourse. He was otherwise temperate. For two or three months preceding the sudden development of the paraplegia, he experienced at times some difficulty in micturition. The urethra was healthy. On the 9th of December there was numbness of the lower extremities extending as high as the knees, but this was so slight as not to attract any attention at the time. On Monday, the 14th, he walked as usual from the suburbs to his business in the City. About the middle of the day, as he was crossing his room, his legs suddenly became weak, and he would have fallen had he not been supported. After a short time he recovered sufficiently to walk with some difficulty to the omnibus, and afterwards from the omnibus to his home. In the course of the afternoon he became entirely paraplegic. the urine and fæces passing involuntarily from him. There was no affection of the upper extremities except slight and transient formication in the hands.

On admission, on the 23rd, there was only a trace of excito-motor activity in the left leg, and none in the right. There was no appreciable diminution of sensation. Movements in the chest normal. Pulse 120, feeble. Pupils dilated. Surface of trunk and upper extremities warm and perspiring. Legs cold. A sense of tightness around the chest, about the attachment of the diaphragm. Bowels inactive. Urine, drawn off by catheter, acid.

The day following his admission there was noticed to be some œdema of the integuments in the lumbar region, especially on the right side. On the 26th this had almost disappeared. The spine was normal. No change in the paralytic symptoms. Occasional slight involuntary twitchings of the legs. *Electro-contractility of the muscles good.* Only the slightest trace of excito-motor action, and that limited to the left leg. The integuments over the sacrum reddened. Pulse 130. Skin hot and dry. Urine ammoniacal, and containing a large quantity of very offensive mucoid pus. The passage of the catheter was followed by much bleeding. During the night of the 28th nausea and vomiting came on, with great prostration. Respiration thoracic. Death from exhaustion on the morning of the 30th, the case having been brought to a rapid termination by the supervention of acute peritonitis upon inflammation of the bladder. The upper extremities were unaffected throughout, with the exception of the slight and transient formication noticed above.

Post-mortem examination.—Head not examined. About the base of both lungs, commencing acute lobular pneumonia. Lung tissue otherwise healthy. Heart healthy. Intestines covered by recent inflammatory exudation. Mucous membrane of bladder sloughing. Its muscular coat, and the pelvic areolar tissue, infiltrated with fetid pus and urine. Two false passages, one passing through the prostate and thence into the bladder, and the other passing into the areolar tissue behind it. No stricture of the urethra. Texture of kidneys healthy. No trace of old or chronic disease could be discovered, either about the pelvis, in the pelvic viscera, or in the bodies of the vertebræ. The larger veins were opened, but afforded no evidence of phlebitis. Integuments over the sacrum beginning to slough, over the lumbar region they were œdematous. Membranes of the cord healthy. As the finger was passed lightly along the body of the cord it appeared to be somewhat softened at two points, in the middle, and at the lower part of the dorsal region; but on the most careful microscopical observation nothing abnormal was discovered in the texture either at these parts or in any other, though the cord was submitted to repeated and searching examination by the microscope. The epithelium lining the ventricle of the cord in the lower dorsal and lumbar regions was abundant, but normal. A few granules of brain-sand were found in the posterior columns, about the middle of the dorsal region. No traces of inflammatory exudation anywhere, either in the cord or in its membranes, nor any evidence of degeneration of the nerve-tubules.

Remarks.—When this patient came under care it was thought that the paraplegia was the result of ramollissement of the substance of the cord, which had (as not unusually happens) been more or less latent in its progress, the sudden paraplegia coming on when the conducting tubules have reached a point of degeneration which destroys their continuity. The examination of the cord did not confirm this diagnosis. In the present state of nervous pathology the case remains unexplained. It is confessedly difficult to establish a negative, but the difficulty was met with unusual care in this case. Hours were spent in the examination of the cord, but with no other result than to show that there was no appreciable lesion of it besides a slight and doubtful softness of the tissue at two points. We may, therefore, certainly conclude that the spinal cord may have its functions impaired and even lost, and that suddenly, as far as the power of motion is concerned, without any distinct amount of anatomical lesion. Some writers have thought that the cord might be paralysed by a morbid impression made upon it, through incident nerves, and independently of any lesion of structure. Mr. Stanley sought to establish this in reference to disease of the kidneys, believing that these organs when congested might, through their nervous connections, set up paraplegia. I have shown in another place ('Med.-Chir. Trans.,' 1854) that the cases recorded by that author do not support his theory. In the instance before us there was no lesion of the kidneys or of the pelvic viscera preceding the paraplegia; nor does there in the history of the case appear to be any sufficient cause for the paralysis, unless we accept it as one of acute tabes dorsalis, resulting from over sexual indulgence. Had the case not been rapidly terminated by cystitis and acute peritonitis, the cord, examined at a more advanced period, would, in all probability, have presented definite degenerative changes; or perhaps it might have recovered itself by the slow processes of nutrition. It is worthy of notice that sensation was not affected. In the treatment, cupping, blisters, and mercury would have been obviously inappropriate. Wine and opium moderately in the beginning, and at a later stage the mineral tonics, were the means indicated; but unfortunately, as too often happens, the accidents of the paraplegia, the pelvic complications, gave no opportunity for the successful issue of the case. In this respect women have the advantage over men, catheterism being less needed, or when required, less liable to produce injury in them than in the male sex.

CASE 18 (Plate II, fig. 1).—*Numbness and weakness of legs for several months; sudden onset of pain and increased debility; no impairment of sensation; temporary increase of voluntary power under the use of strychnia, soon followed by complete paraplegia; retro-peritoneal abscess between bladder and uterus; death from peritonitis; remark-*

able atrophy of the grey substance of the cord; chronic inflammatory degeneration of the posterior columns.

(Reported by Mr. DURHAM.)

Harriet B—, æt. 50 (?), but looking much older. A widow employed as a nurse. Admitted into the hospital 10th December, 1857. Seven weeks before this she was suddenly seized with acute pain in the right foot, so severe that she could not move the leg. In a few days the left foot was similarly affected. The pain gradually subsided, but only to return at intervals as severely as ever. The muscular power became at the same time impaired. She could move the limbs when lying down, but not leave her bed. On questioning her it appeared she had for some time felt uncomfortable sensations in the legs, with slight numbness, and a feeling of debility, but was able to perform her duties until the time of the sudden seizure of pain. When she came into the hospital there was only just sufficient voluntary power over the legs to flex them slightly, the left rather the most. Occasional feeble involuntary jactitations, and distinct but not very marked excito-motor movements. Sensation not impaired. Urine drawn off by catheter, ammoniacal and containing mucus. Pain over the abdomen; occasional vomiting. Strychnia was given in doses of $\frac{1}{16}$ grain. Under its use the voluntary power was for a few days rapidly increased, but at the end of a week the spasms of the legs were so violent the medicine could not be continued. The cord was left exhausted, and, at the end of five weeks after her admission, the legs were completely paralysed, and no excito-motor movements could be produced. Sensation now seemed to be impaired, but the patient at this period of her illness lay for the most part in such a dull and stupid state that it was difficult to form a satisfactory opinion on certain points. The skin was abraded over the sacrum and trochanters. Highly offensive urine dribbled from the bladder. She would not submit to have anything done with the catheter, on account of the pain it caused her. She lay in a state of semi-coma, and died exhausted, February 5th, 1858.

Post-mortem examination.—Body wasted. Head not examined. Spinal bones, ligaments, and theca vertebralis, healthy. Arachnoid normal, with one or two fibroid plates on the visceral layer. Spinal cord in the lower dorsal region small, and soft to the touch; the anterior fissure gaped open. Examined in the fresh state, abundant granule masses (exudation cells, &c.), having the usual appearance, were found in the columns. On section, the centre of the cord formed an irregular depression from atrophy of the grey substance. These changes were, however, more definite after the cord was hardened by immersion in spirit and thin sections made of it. The atrophy affected the fibrous portion of the grey substance. The caudate vesicles had their normal position and structure. There was no exudation amongst the grey substance. The symmetry of the changes in the columns, and the mode of extension of chronic disease in them, are well shown in Plate II, fig. 1. The lesion was due to chronic inflammation and concomitant atrophy of the tissue, with subsequent fatty degeneration of the newly effused matter. The fatty incrustation of the capillaries was a mechanical result, as shown in Case 21. Cortical portions of both kidneys full of points of suppuration.

Pelves, ureters, and bladder, acutely inflamed. A large retro-peritoneal abscess between the bladder and uterus, but not communicating with the bladder. Recent inflammatory effusion over several coils of intestines in the pelvis. Viscera of chest healthy.

Remarks.—An inspection of the section given in Plate II, fig. 1, will show how much could be expected from treatment. It cannot be objected that the lesion there depicted does not convey a true impression of what existed during life, since there is no evidence of recent changes. It is an important consideration in the treatment of diseases of the nervous centres, how far the symptoms are due to irremediable changes or not. A lesion of the nervous tissue may be cured—or, at least, be in a state which, if it were in the skin or muscle or gland, would be called cured—and yet, according to the patient's estimation, the disease may remain. We too often think of symptoms as substantially the disease; and if this false view guides our treatment we cannot fail of doing harm. We waste the feeble powers of an already partially dilapidated system, instead of recognising the dilapidation as an essential and permanent condition of the body we have to treat.

The therapeutical agency of strychnia in organic lesions of the cord has yet to be proved. Judging from its effects, we should say its direct operation on the tissue was the very reverse of nutritive or reparative. If function is, as there can be no doubt, the effect of a mode of disintegration, agents which directly increase function must produce a disintegrating action. If this be a sound inference—and experience leads to the same conclusion—strychnia has but a limited therapeutic application in paraplegic affections. It is well known that immediate and striking effects can be produced by this drug, but these are often followed by hopeless bankruptcy of the spinal power. In giving strychnia our object should be to produce no greater change of the tissue than shall, by the stimulus of waste, increase the power of nutrition, as we exercise an organ to favour its healthier growth. This requires not only a diagnosis of the conditions producing the paraplegia, but a careful adaptation of the dose of the medicine, which is often a difficult point. I have seen one twenty-fourth of a grain given twice a day for only two or three days in a case of chronic paraplegia, apparently depending upon softening of the cord, set up very decided irritation. So unstable is the structure of the nervous tissue in some of these cases, and so delicate in proportion must be our interference by remedial agents.

CASE 19 (Plate II, figs. 2 and 3).—*Chronic inflammatory degeneration of the posterior columns of the cord throughout their whole length; the disease strictly limited to the posterior columns; frequent vomiting; general emaciation of the voluntary muscles; paraplegic weakness of lower extremities, characterised by a want of control over the contraction of the muscles; congenital misplacement of the ascending colon, which became twisted on itself; cæcum sloughing.*

William J—, æt. 28, of middle stature, fair hair, emaciated, anxious expression, large head, broad and prominent forehead. Though he had never been robust he had good health until the beginning of the year 1857. He was first seized with vomiting, which came on without any discoverable cause, and lasted for several days. As he recovered from the attack, the legs became weak. After three months he had a second attack of vomiting, followed by an increase of weakness in the legs. He was admitted into Guy's Hospital under my care, November 11th, 1857. He was then unable to stand without support. In a recumbent position he could flex and extend the legs with some freedom, but the movements were sudden and vague from want of control over the action of the muscles; the spinal centres, when stimulated by the will, seeming to shoot off their influence at once, making the feeble muscles contract to their full extent with a jerk. In other words, there was no power to regulate the muscular contraction. The movements of the fingers were also wanting in precision. He was awkward in handling small objects or in applying the hand to grasp larger ones. The muscles were thin and flaccid, corresponding to the general emaciation of the body. The muscular irritability was excessive. Weak currents of electricity, not sufficient to affect healthy muscles, excited well-marked contraction; whilst a little stronger but yet very moderate dose of electro-magnetism produced cramps lasting for several seconds after the stimulus was discontinued. The arms were weak, with an obvious want of control over the voluntary movements. There was numbness of the feet and hands, and a burning formication in the fingers and toes. The sensation of the other parts of the body was normal. No involuntary contraction of the legs. Sphincters good. Urine acid. The lower ribs depressed, and but little moved in inspiration. Headache, vertigo, cerebral confusion, tinnitus. Pupils largely dilated, the left the most so, sight dim, occasionally transient amaurosis. Sleep disturbed by dreams. Frequent nausea and vomiting, with pain from the epigastrium to the spine. Abdomen not distended, soft. Skin hot and perspiring. Pulse permanently quick, 126, small and feeble. Respiration 32. Spine straight. No tenderness on pressure or percussion.

He could give no account of any accident or injury to the spine, except such as might have resulted from a fall, flat on the back, from the height of a few feet, eight years before his symptoms began. His habits had been temperate. No syphilitic taint.

After admission into the hospital he continued to have repeated attacks of vomiting, lasting for many days, uninfluenced by any remedies. The vomited matters were copious, greenish, and mucous. The bowels continued to act freely, but without relief to the sickness. The irritability of the stomach was attributed to the state of the cord. The attacks of vomiting increased his anxious aspect. The paraplegic symptoms continued unchanged. There was, as before noted, headache and vertigo, and sometimes transient amaurosis. The pupils remained permanently dilated, and with the same inequality. The pulse quick (120 to 130) and feeble. On only one occasion was the urine noticed to be alkaline when first passed. It never contained mucus. There was no band-like sensation around the abdomen. He often complained of pain from the epigastrium through to the spine. About the middle of February, 1858, he first had a sense of bearing down about the rectum, and complained of great distress after an action of the bowels, and of startings in the legs. March 8th he had an attack of vomiting, apparently such as had often occurred before. This continued on the 9th. On the 10th he was collapsed and pulseless, with cold sweats, and other symptoms of ruptured intestine. There was no cerebral oppression. He died on the 11th.

Post-mortem examination.—Body emaciated. Brain healthy. Thoracic viscera healthy. Fæcal extravasation into peritoneal cavity. Intestines adherent by recent lymph. Omentum contracted into a cord-like mass, and firmly adherent to the left side over the pubes. Cæcum fallen into the cavity of the pelvis. From it the ascending colon passed directly to the left side towards the spleen, and then curved down again before becoming continuous with the descending colon. This displacement of the cæcum and ascending colon arose from a congenital (?) absence of the mesocolon on the right side. The ascending colon, at its commencement, was partially twisted upon itself. Both it and the cæcum were dark coloured, and sloughing to a large extent from mechanical obstruction. The spinal cord had its normal appearance and consistence, except, perhaps, a small portion in the dorsal region, which seemed rather softened; but this was doubtful, and was only such as an accidental tension in moving it from the canal might have produced. Sections of the cord made at the time gave no further evidence of disease. The membranes were healthy. After hardening the cord and making fine sections it was seen that the posterior columns were atrophied throughout their whole length, and amongst the tissue were numerous exudation cells in a state of fatty degeneration (granule cells). The posterior roots and the lateral columns were normal (Plate II, figs. 2 and 3). The disease was limited above by the commencement of the medulla oblongata. No degeneration of structure had occurred in this part.

Remarks.—The relation of morbid conditions to each other is often difficult to determine. It was so in this case.

The oldest disease was, no doubt, that discovered in the abdomen; the absence of the mesocolon on the right side was evidently congenital, and probably the adhesion of the great omentum dated also from an early period. It was these lesions which brought about the fatal result. The steps of the process may be looked at in two ways. It may be admitted that a cæcum and colon left to float free were in danger of getting into positions unfavorable to the propulsion of their contents, and thus of occasioning attacks of vomiting, such as ushered in the other symptoms in this case, and continued to harass and distress the patient throughout his illness. It may also be thought probable that attacks of abdominal disturbance might, through incident nerves, set up a secondary lesion in the cord. This, however, is doubtful, and it is more in accordance with our pathological views to refer the early attacks of vomiting to the lesion of the cord itself as the primary disturbance, especially since the degeneration of the columns extended up to the neighbourhood of the medulla oblongata. With this view it is not difficult to understand how the congenital defect in the colon should be brought into fatal operation by irregular peristaltic action so induced.

The limitation of the disease to the posterior columns was remarkable (Plate II, figs. 2 and 3). Though they were degenerated throughout their whole length from the lumbar portion to the medulla oblongata, neither the posterior roots of the nerves nor the adjacent parts of the lateral columns were in any way involved in the degeneration. We might, from this strict limitation of the lesion, hope to gain some unequivocal evidence as to the physiology of these structures.

The affection of sensation was limited to numbness and formication of the hands and feet. Dr. Brown-Séquard has shown, by transverse section of the posterior columns in animals, and by instances of disease in the human subject, that where the posterior columns are destroyed for a limited extent, as by pressure of a tumour, hyperæsthesia is produced in the parts below the injury; in both extremities if the lesion affects both columns, but only on the side of the lesion if one column is affected. When, however, the posterior columns are destroyed throughout their whole length, instead of hyperæsthesia, there is loss of sensibility to some degree. Not that the posterior columns convey, according to this observer, sensitive impressions to the brain, but because, being in part channels through which the fibres of the posterior roots reach the grey matter, if they are destroyed or degenerated throughout, a certain number of sensitive fibres must be destroyed also. So far theory coincides with the facts noticed in this case.

The same physiologist believes that the special function of the posterior columns is for the reflex movements. These functions ought, therefore, to have been destroyed, or at least greatly diminished. Nothing in favour of such a theory was, however, noticed, except the general muscular emaciation.

The sphincters of the rectum and bladder continued to perform their office. The muscles remained in a state of hyper-excitability to the galvanic stimulus. I do not know how far the state of the colon might be referable to a loss of the reflex power.

Neither were the phenomena more in favour of the value of the test proposed by Dr. Marshall Hall, for the diagnosis of cerebral from spinal paralysis, since, according to that, the irritability of the muscles should have been much diminished, the disease being in the cord ; but, on the contrary, it was remarkably increased.

This brings us to the theory of the posterior columns proposed by Dr. Todd, that they "propagate the influence of that part of the encephalon which combines with the nerves of volition to regulate the locomotive powers, and serve as commissures in harmonising the actions of the several segments of the cord." The want of power in this case to regulate the action of the muscles was very characteristic. The legs, when drawn up, as they could be freely, were drawn up with a sudden jerk, and extended in the same manner. The voluntary movements of the hands were also fumbling and vague.

The limitation of the disease to the posterior columns coincides with what is generally found. There is evidently a tendency in lesions to spread longitudinally in the cord rather than transversely through it,—probably from homogeneity of structure or from the arrangement of the blood-vessels.

Such complete and symmetrical isolation of a structure is very suggestive of an independent function.

It is unnecessary to refer particularly to the character of the morbid changes in this case. They were evidently of that kind which we denominate by the term "chronic inflammation,"—atrophy of the proper tissue, with exudation, which corpusculates and then becomes fatty. This change was probably induced by the fall on the back eight years previously.

In the ordinary mode of examination the disease of the cord in this case must have been overlooked, and it would probably have been regarded as one due to cerebral disease, though certainly there was no evidence of it post mortem. Clinically, there was more to support such a view, namely, headache, vertigo, cerebral confusion, tinnitus, dilated pupils (one larger than the other), dim vision, occasionally transient amaurosis, sleep disturbed by dreams, &c. These symptoms —together with power to move the limbs when in a recumbent posture, but inability to stand without support, and apparently a great increase of all the symptoms when the patient is in a vertical position—led Dr. Baillie to assume that the seat of the disease in these cases is in the encephalon. In the year 1848 I proposed a classification of paraplegia which should recognise the existence of such cases; but a better method of investigating the morbid changes in the cord daily lessens the number of instances referable to such a division, and makes it doubtful whether paraplegia properly so called is ever due to lesions which are strictly cerebral in their seat.

CASE 20.—*Paralysis of both seventh nerves; nearly complete paraplegia of lower extremities; weakness of upper extremities; onset of symptoms acute; death on the ninth day; for some months preceding the invasion of the paraplegic symptoms pains in the left arm and slight wasting of the muscles, supposed to be rheumatic; wasting of the grey commissure on the left side of the cord in the cervical region; recent inflammatory exudation into the tissue of the medulla oblongata and into the grey commissure of the cord.*

Mr. E—, æt. 59, began to suffer from pain in the left arm, from the shoulder to the elbow, at the end of the year 1856. The pain continued some months, and left the arm weak and slightly wasted. This was regarded as a rheumatic affection. There was no anæsthesia; Mr. E— was in other respects in good health until Christmas, 1858. He could give no more precise description of the beginning of his indisposition than that he was languid. He spoke also of an occasional feeling of coldness between the shoulders and down the spine, attended with distressing rigors in the spinal muscles. At the beginning of March, 1858, he had bilious vomiting, with pain in the right hypochondrium. He was able to continue the active duties of his profession as a medical practitioner, and gave a public lecture on the evening of the 29th of March, but said that he felt more weak and tremulous on that day than usual. He visited his patients on the 30th, and appeared in his ordinary health, but in the evening complained of constricting pains in both arms, from the shoulders to the insertion of the deltoid muscles. On rising from bed on the morning of the 31st he found his legs too weak to support him, and from that time his paraplegic symptoms rapidly increased. I visited him on the 4th of April. There was then entire inability to move the muscles of expression on either side of the face. The involuntary action of the orbiculares palpebrarum continued, but the eyes could be only partially closed by volition. The features hung motionless. He first noticed the paralysis of the face the day previous, when attempting to put up his lips to kiss his wife. The motor and sensitive divisions of the fifth nerves were unaffected, except slight anæsthesia of the first division on the left side. Motions of the eyes, vision, hearing, taste, and deglutition normal. Respiration chiefly abdominal, the movements of the lower ribs being defective. Numbness of the fingers of both hands. Movements of upper extremities free. He lay supine with the legs extended and powerless. Muscles flaccid. No excito-motor movements on irritating the soles of the feet; sensation impaired, and a feeling as of a board pressed against them. The sphincters retained their power. Urine pale straw-colour, acid. Intellect perfectly clear. Tongue protruded straight. Articulation perfect for all words not requiring the use of the lips. Labials could not be pronounced. Pulse 72. Respiration tranquil. On closer inquiry as to any premonitory symptoms it was elicited that in the summer of 1857, on one occasion in sleep, an evacuation had passed from him involuntarily. This was the only evidence of spinal disorder except that given above. On the 6th there was a slight return of power over the muscles of expression. The paraplegic symptoms, however, were unchanged. On the 8th the respiration was more feeble, and chiefly abdominal. Tongue dry and brown. Dribbling of urine. When roused he was quite collected, but left to himself there was wandering delirium. Movements of arms very feeble; slight subsultus. He died in the evening very tranquilly, the breathing ceasing so gradually that the last respiration could not be told.

Post-mortem examination.—Rigor mortis well marked, both in upper and lower extremities. Large amount of subcutaneous fat over chest and abdomen. Muscles of lower extremities well developed. Left arm slightly less muscular than right. Large deposit of fat about the base of the heart and over the right ventricle; valves healthy; aorta extensively atheromatous. Lungs healthy. Universal, old, tough adhesions between the diaphragm and upper surface of liver. No corresponding adhesion of the pleura above. Kidneys large, tunics easily stripped off, surface smooth. Bladder healthy. The convolutions of the hemispheres of the brain separated by clear subarachnoid effusion. No other abnormal change. Corpora striata, thalami optici, cerebellum, and pons Varolii healthy. The basilar and vertebral arteries opaque and rigid. The trunks of all the cerebral nerves healthy. Bones, ligaments, and membranes of the spine healthy. In the cervical and dorsal regions the substance of the cord was to the touch somewhat softer than natural, but no other unequivocal change was discoverable by the unassisted eye, or by the aid of a common lens. On hardening the pons Varolii, medulla oblongata, and cord, and preparing sections after a modification of Lockhardt Clarke's method, it was seen that in the anterior part of the commissure, throughout the length of the cord, but principally in the lumbar and superior cervical regions, and throughout the structure of the medulla oblongata, but chiefly at its superior part, there were exudation cells scattered interstitially amongst the tissue; they were also seen, but more sparingly, in the lines of areolar tissue which radiate through the white substance and amongst the deeper part of that which dips into the anterior commissure. There was no want of continuity nor any destruction of the nervous tissue. The cells were recent, and had not undergone fatty degeneration. The amount of the exudation was so small and its distribution such that no lesion was visible, except under the higher powers of the microscope. It was then very distinct, and remains so in the sections preserved in Canada balsam. In the cervical region on the left side there was wasting of the grey commissure and a development of fibrous tissue in its place.

Remarks.—The supposed rheumatic affection of the left arm was referable to the changes in the grey commissure in the cervical region. This change was very limited in extent, but still very definite when transparent sections of the cord were examined. The experiments of Brown-Séquard—which go to prove that injury to the grey matter of the cord on one side alters the sensibility on the opposite side of the body—seem opposed to the facts in this case. It is probable, however, that the painful affection of the left arm was due to a lesion of the motor nerves—the chronic changes in the muscles subjecting the textures to unnatural tension.

The paralysis of both seventh nerves was a striking incident in the case. It was referable to the central changes which extended through the tissue of the medulla oblongata. The nerve-trunks and surrounding parts were healthy. The exudation estimated in mass was very trifling, not sufficient, indeed, to give unequivocal evidence of its presence but for our improved methods of research. It is not, however, to be forgotten that its seat was in the most important part of the nervous centres, where nature has afforded no surplusage.

The defective speech led those about the patient to suppose the symptoms were due to disease of the brain. It, however, needed but little investigation to show that this defect was entirely due to paralysis of the lips, and was limited to the pronunciation of labials, other parts of speech being pronounced distinctly.

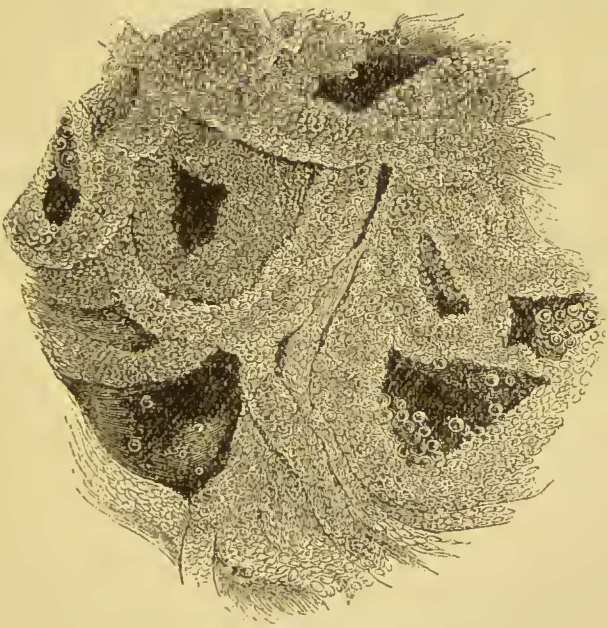
The intellect was undisturbed. The patient gave a very clear account of himself. The tongue was moved freely. Digestion unimpaired, and the breathing natural.

The diagnosis was of softening of the cord, but strictly speaking this was not the lesion. It was an inflammatory exudation into the more vascular parts of the cord and medulla oblongata. To what condition of the circulating fluids or of the blood-vessels this was attributable is conjectural. The patient was a beer and porter drinker, his subcutaneous tissues were loaded with fat, his age fifty-nine,—conditions which are associated with and favour a gouty state no doubt much oftener than the occurrence of distinct gout would seem to indicate, for a patient may be gouty who has never had gout, as one may be poisoned by marsh miasm who has never had ague. There was no history of injury or of exposure to cold. The effusion under the cerebral arachnoid was probably the result of that capillary paralysis (congestion) which comes on in death from disease of the nervous centres.

CASE 21.—*Sudden paralysis of right leg; partial recovery after five months' acute paraplegia; erysipelas and consecutive pneumonia from the application of heat to the legs; irritative fever, and death in two weeks; recent softening of the cord in the dorsal region; old degeneration of the right lateral column, with fatty incrustation of the capillaries.*

Ed. M—, æt. 34, a man of dissolute habits, but originally of a strong and well-developed constitution. At the end of November, 1853, on rising from his bed felt himself suddenly powerless in the right leg. He had at the same time pain in the lumbar region, extending to the hypochondria.

There was no anæsthesia. He asserted that he had felt nothing wrong with himself previously; and, so far as he knows, there had been no premonitory symptoms of the paralysis. For more than a month the leg remained completely paralysed, "there was not the slightest power of motion in it." Pain in the course of the sciatic nerve. Urine drawn off by the catheter, ammoniacal. Frequent priapism with spermatic discharges (as proved by microscopic examination), but he was not himself aware of any excitement of the genital organs when questioned about it. He was treated by Mr. William Hills with laxatives, the preparations of iron and strychnia, and by galvanism. Slowly the pain left him, and he recovered some power over the leg so as to be able to stand upon it, and to walk with the aid of a stick, but he could not flex the muscles of the hip-joint. With this improvement he relapsed into his former habits, and after a week rather suddenly became paraplegic. He was admitted into the hospital April 29th, 1854. The right leg was then wasted and completely paralysed. Slight power remained in the left leg, and there was frequent involuntary jactitation of it. It was swollen from erysipelatous inflammation which had extended from a bulla caused by the application of a hot bottle. Irritative fever followed, and death at the end of a fortnight from the time of the relapse.



Sketch of capillaries incrusted with oil-globules, Case 21.

Post-mortem examination.—On the left leg superficial excoriations and the remains of blebs. Subcutaneous cellular tissue containing collections of pus. Saphena vein not implicated. Bones, ligaments, and membranes of spinal cord healthy. Large quantity of transparent cerebro-spinal fluid. In the upper dorsal region the substance of the cord was softened to the extent of an inch; exudation-cells scattered through the tissue. In the right lateral column, near the same part, the tissue was atrophied and the capillaries incrusted with oil-globules (*see* Fig., p. 225).

The fatty matter could be removed by ether, leaving the walls of the vessels apparently normal. This change was strictly limited to the right side of the cord. Recent lymph on lower lobe of right lung. Pneumonic consolidation of the bases and posterior parts of both lungs. Liver pale, weight 4 lbs. 3 ozs. Kidneys congested, weight 12 oz. Mucous membrane of bladder thickened, congested, and greenish.

Remarks.—The chief pathological interest of this case lies in the suddenness of the paralysis in the first instance, and its limitation to the right leg. The cause of this was plainly made out on examination of the cord. The incrustation of the capillaries with oil-globules appeared to be nothing more than a mechanical result, and not due to a degeneration of the coats of the vessels. In pathological changes of the nervous substance we may distinguish these two conditions. In the one the changes in the capillaries are probably antecedent to the lesion of the textures, and in the other consecutive to it.

It is a matter of speculation what set up the softening. Dissolute habits induce many conditions predisposing to such a change; and amongst them, perhaps, none more efficient than the contamination of the syphilitic virus. In softening of the brain there can be no doubt of this connection, and that, too, apart from any noticeable cachexia.

The seat of the softening corresponded with that so frequently found in other cases. The dorsal region, from its position and organisation, is exposed to lesion, and the cord has, perhaps, at this part, less resisting power than at other parts which are more highly organised.

CASE 22.—*Paraplegia supervening two days after a violent exertion in lifting a heavy weight; softening of the cord opposite the fifth and sixth dorsal vertebræ; no injury of the membranes, ligaments, or bones of the spine; death after six weeks.*

Richard A—, æt. 25, of a rather delicate constitution, was at his usual occupation as a labourer in the Commercial Docks on Saturday, November 22nd, 1856, when, after lifting some deals, he felt a sudden pain in the back. He walked to his home, the distance of a mile and a half, and the following day was apparently quite well. The next morning (Monday), on waking, the legs were paralysed. When admitted into the hospital, November 26th, there was complete paraplegia, a bedsore had already begun to form over the sacrum, and ammoniacal urine dribbled from the bladder. He died exhausted, January 2nd, 1857.

Post-mortem examination.—Body emaciated; large bedsore exposing the whole length of the sacrum. The bones and ligaments of the spine in the other regions were carefully examined, but no trace of injury was discovered. Opposite the fifth and sixth dorsal vertebræ the cord was softened through all the columns into a thick, greenish, muco-puriform fluid, with a tinge of brown. Examined by the microscope, it was seen to consist of disintegrated nerve-tissue, with a few irregular collections of granules. *The cord was not enlarged at the softened part, nor was there any trace of inflammatory exudation in it or upon the membranes covering it*, though to the unassisted eye it had the appearance of an irregular undefined abscess. The lumbar and cervical portions of the cord had the normal appearance and firmness. A large portion of the lower lobe of the right lung and half the upper lobe of the left were hepatised. Heart normal. Liver large and fatty. Commencing suppuration in the cortical substance of the kidneys. Mucous membrane of the pelves greenish, with patches of adherent fibrinous exudation. This condition of the mucous membrane was continued through the ureters into the bladder. The bladder contained a quantity of muco-purulent fluid.

Remarks.—This case shows that the substance of the cord may receive an injury through violent muscular exertion, whilst the surrounding textures escape. Why this should rather occur in the dorsal region is obvious, since the curve of the column is most marked and most variable, and the body of the cord is thinnest, at this part. It is a matter also of common clinical experience that the cord is very prone to softening in the dorsal region, from which we may, perhaps, infer that, in addition to its being here more subject to injury, it has a more feeble organisation than the cervical and lumbar enlargements. The change in the cord was seen by the microscope to be due to mere disintegration. There was no evidence of any plastic exudation. The greenish and brownish tints of the softened part were probably due to blood-colouring matter. We may infer, from the quality of the local changes, which appear to have been quite passive, and from this slight coloration, that the immediate effect of the injury was upon the capillary circulation, leading to effusion of blood and consequent atrophy.

Case 23.—*Concussion of the cord in the cervical region from direct violence; ecchymosis into posterior horn of grey matter on left side, also into anterior horn on right side and into the posterior columns; loss of sensation immediately after the accident, followed by hyperæsthesia; paralysis of legs, left arm, and sphincters; death thirty-four hours from the accident.*

(Reported by Mr. Bankart.)

Joseph K—, æt. 33, a coal-porter, strong and healthy, was carrying a sack of coals on his back, down some cellar stairs, when his foot slipped forwards from under him and he fell, the sack of coals falling upon him. On his admission, immediately after the accident, 3 p.m., June 22nd, 1858, there was loss of motion of both legs and of the left arm. The sphincters were paralysed. There was entire loss of sensation in the left arm as high as the deltoid. The right arm he could move, and had perfect sensation in it. On examining the state of sensation in the lower extremities, it was found that he could feel about the feet and on the outer side of thighs, but not on the anterior and inner surface. During the time marks were being made on the skin to indicate the state of the sensation at different parts, it was found to vary, returning to spots where it had just previously been absent. Apparently the most distant parts recovered first. Slight priapism. Breathing diaphragmatic. After a few hours sensation returned in every part. As the skin became warm he complained of pain when lightly touched. For instance, when the finger-nail was passed but lightly along the skin he would exclaim, "Don't prick me! don't hurt me!" The day following, the sensibility of the surface appeared to be excessive, judging by his exclamations when the skin was touched or pinched. This was especially noticed in the right arm. Priapism, which existed when he was admitted, passed off after two hours, but returned the day following. He continued to have power to move the right arm. He died thirty-four hours from the accident.

Post-mortem examination.—The spine only was examined. There was no external trace of the injury; no displacement of the vertebræ discoverable by external examination. The membranes of the cord were healthy. Opposite the fourth and fifth cervical vertebræ the substance of the cord was contused. On section there was found ecchymosis of the posterior horn of grey matter on the left side, and of the adjacent part of the lateral and posterior columns. There were also other limited spots of ecchymosis on the right side, one in the right posterior column, and one in the anterior cornua of the grey substance. The grey matter generally was hyperæmic (from venous congestion?), but there was no other lesion of it except at the two spots named; no lesion of the anterior columns. The commissure was uninjured. On examining the spinal canal after the removal of the cord, nothing abnormal was discoverable in the bodies of the vertebræ opposite the lesion of the cord; but on dissecting off the posterior ligament it was seen that the body of the fourth was separated from that of the fifth, and that the left articular process of the fourth had been chipped off by the violent pressure of the lower one against it.

Remarks.—There are several points worthy of note in this case; the character of the injury received by the cord, namely, limited capillary ecchymosis; the absence of any external sign of the injury; the mode by which the cord was injured, namely, by concussion, and not by pressure of surrounding parts upon it, as shown by the ecchymosis being in the substance of the cord, whilst its peripheral parts and membranes had escaped; the limitation of the injury, producing paralysis of the left arm, whilst the right retained the power of motion; the immediate effects of the concussion on the cord, producing anæsthesia for a few hours; the return on sensibility first in the parts most distant from the injury, and the development of hyperæsthesia. This latter symptom was in accordance with the experiments of Séquard, who has shown that injury of the posterior cornua of the grey matter is followed by hyperæsthesia of parts below. Cases of injury, as before remarked, have as much interest to the physician as to the surgeon, since they often come under the care of the physician for the treatment of the permanent effects; when it is necessary there should be a correct estimate of the character of the primary lesion.

CASE 24.—*Concussion of the cord by a fall; recovery of power after two hours; subsequent effusion of blood outside the theca vertebralis in the neck; paraplegia of upper and lower extremities; paralysis of intercostals; intense heat of skin; death in fifty-five hours.*

(Reported by Mr. VENOUR.)

Robert L—, æt. 40, fell backwards from a moderate height, a heavy plank falling at the same time upon him. He was at once brought to the hospital (4 p.m., July 7th, 1858). He was collapsed, but sensible. There was entire paralysis of the left leg, partial of the right, and also partial paralysis of the arms, but he was still able to flex the fingers. After two hours he had so far recovered from the immediate effects of the injury that he could draw up his legs and grasp the hand; the circulation was improved surface warmer. No injury of spine discoverable. At 10 p.m. he said he felt comfortable. He passed a restless night, and the following morning, at 8 a.m., was entirely paraplegic both in the upper and lower extremities. Loss of sensation in the paralysed parts. Priapism. Ribs scarcely moved in inspiration. Temperature of surface increased. Abdomen tense and tympanitic. During the day the skin became intensely hot, but the actual temperature was not noted. The breathing was wholly diaphragmatic. Deglutition difficult. He died fifty-five hours from the accident.

Post-mortem examination by Mr. Bryant.—No external evidence of injury to the spine. On dividing the soft parts there was found a separation between the fourth and fifth cervical spinous processes, and dislocation of the articular processes. The interspinous and capsular ligaments were torn through. Extravasation of blood outside the theca vertebralis on its anterior aspect. The effused blood compressed the cord, which was otherwise uninjured. After careful examination there were not found any signs of bruising of its tissue. The extravasation apparently arose from injury to the lower part of the body of the fourth vertebra, which had been fractured, and the intervertebral substance torn. The calibre of the canal was slightly encroached upon by displacement of the fourth vertebra, but not so as to press on the cord. The extravasation, though most abundant opposite the injury, extended downwards to some distance. The membranes of the cord were uninjured.

CASE 25.—*Cervical paraplegia following an injury; progressive muscular atrophy of the upper extremities, most marked on the side of the principal lesion in the cord; anæsthesia, with severe neuralgic pains on the opposite side; paroxysms of hiccup for several months; thickening and adhesions of the membranes of the cord; degeneration of the posterior columns; dilatation of the ventricle of the cord; opacity and fatty degeneration of the arachnoid of the brain; ependyma of ventricles granular.*

John G—, æt. 49, a coal waggoner, was forced backwards from his seat by striking his head against a beam whilst driving under an archway. Several ribs were fractured on the left side. Some months after this accident he began to suffer pain from the occiput down over the shoulders, and in about a year the muscles of the upper extremities began to waste. After two years incontinence of urine gradually came on. He was admitted into Guy's Hospital February 11th, 1857, three years from the time of the accident. He then presented a remarkable example of muscular atrophy without actual paralysis. The upper extremities were principally affected. The extensors of the right hand, the muscles of the thumb, and the interossei were extremely wasted. The wrist dropped. The muscles of the shoulder and arm, including the pectoralis major and minor, much wasted, but in a marked degree less so than those of the forearm and hand. Very slight diminution of sensation. He could still lift the arm over the head. The left arm was similarly but less affected than the right, so far as regards muscular atrophy, but there was numbness through the whole arm down to the fingers, and he suffered severely from neuralgic pains in it, which greatly depressed him, and which he described as a compound of smarting and numbness. The trapezii, serrati postici superiores, rhomboidei, and all the long muscles of the neck and back, were remarkably atrophied. The spinous processes were very prominent. No deformity nor tenderness on pressure at any point. The intercostals were so weak that the only respiratory movement was through the diaphragm. The supra-spinati were atrophied, but not to the same extent as the infra-spinati and levatores anguli scapulæ. The legs were wasted and weak, but he was able to walk. Sphincters weak. Dribbling of urine. Constipation. The thorax looked narrow and ill developed from the wasting of the pectorals, the intercostals, and erectores spinæ muscles. The muscles at the back of the neck and the sterno-mastoids were so weak that the head could not be supported erect. Sight dim, drooping of left eyelid. Frequent hiccup for many months. After his admission his principal complaint was of pain in the left arm from the clavicle to the fingers. He described it as a severe smarting with a sense of numbness. His distress from this cause was very great. At the early part of March febrile symptoms set in. Tongue became dry and brown. Frequent hiccup and vomiting. Pain in left arm severe. Dyspnœa. Died March 25th, 1857.

Post-mortem examination.—The arachnoid of the brain opalescent, with spots of white mottling of the more opaque parts from fatty degeneration. Subarachnoid fluid in excess. Ependyma of lateral and fourth ventricles granular, in the latter extremely so. The dura mater on the posterior surface of the cord much thickened. The two layers of arachnoid adherent in patches along this surface, and much thickened by effusion of lymph of old date. Sections of the cord examined with the naked eye gave no distinct evidence of disease. There was a slight yellowishness of the posterior columus, and increased vascularity and thickening of the pia mater covering them. In these columns, especially in the right one, abundance of granule-cells were discovered by the microscope. The exudation was greatest in the middle and lower third of the cervical enlargement. The grey matter was hyperæmic. No exudation into its tissue, nor into the anterior columns. The ventricle of the cord enlarged and distended with delicate granular nuclei. The affection of the cord appeared to be secondary to chronic inflammation of the membranes, and to chronic changes in the ependyma of the ventricle in common with the ependyma of the fourth and lateral ventricles of the brain. Hypostatic engorgement of both lungs, several lobules consolidated from recent pneumonia, some greyish. Other organs healthy.

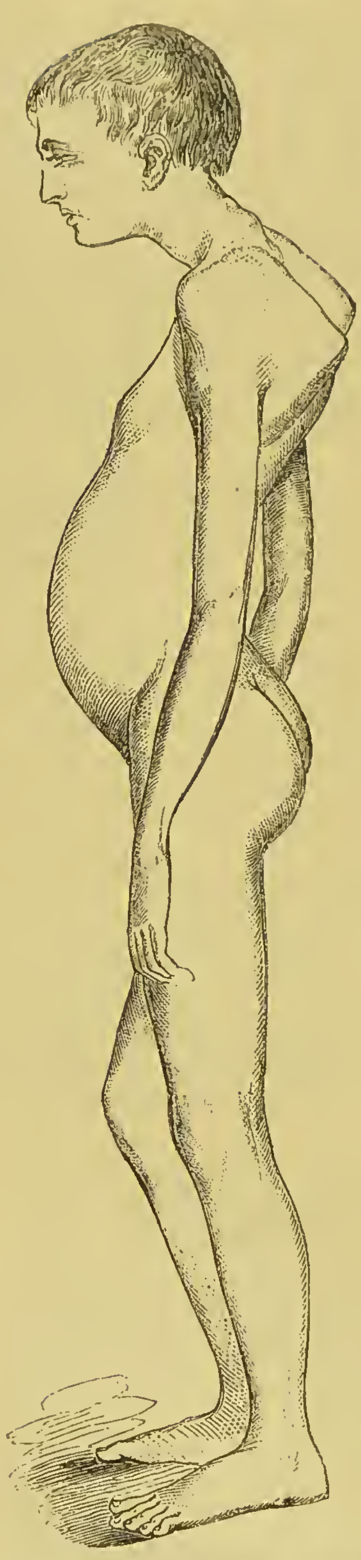
CASE 26.—*Progressive atrophy of the muscles of the trunk and upper extremities after a blow on the neck with the fist.*

Daniel C—, æt. 15, received a blow with the fist between the shoulders from a boy at play. After a week the head drooped, and gradually from that time the muscles of the upper extremities wasted, the arms dropped and hung useless, the intercostals lost their power, and the breathing was diaphragmatic; the lower two thirds of the trapezii and the erector spinæ muscles also wasted in the same way. This sketch was made fourteen months from the injury to exhibit the wasted condition of the muscles and the position of the head and trunk; the head fallen forwards and the trunk thrown backwards to balance it, in the absence of muscular power.

The flattening of the ribs from the paralysis of the intercostals was such that the heart beat to the right of the left nipple and between the third and fourth ribs.

The patient was able to walk about when the sketch was taken. His gait was vacillating, but apparently more from want of muscular power to fix the trunk on the pelvis than from defective power in the legs. He could not sit on a seat without a support to the back. Sphincters good. On testing the electro-contractility of the wasted muscles by galvanism they were found to contract in proportion to their mass; those muscles of the upper arm which were the less wasted contracted well; those of the forearm and hand which were the more wasted contracted less but still distinctly. The progress of the disease was unattended with any pain. The wasted muscles not tender. No flickering contractions of their fibres.

Remarks.—This case is recorded as a good illustration of progressive muscular atrophy after concussion of the cord. It is to be observed that there was no more paralysis than was due to atrophy of the muscles, and that the electro-contractility of the muscles was in proportion to their bulk. These facts are of importance, since it has been erroneously proposed to determine by the test of galvanism the diagnosis between progressive muscular atrophy from morbid changes primarily in the muscles, and that muscular wasting which is consecutive to disease of the cord. It is said that, in the latter case, the muscles early



Sketch showing wasting of muscles after a blow on the neck.

lose their electro-contractility, a statement at variance with extended clinical observation, and further illustrated in Case 19. No doubt, as the lesion of the cord advances in this case (which is still under treatment), the lower extremities will undergo the same changes as the upper. A precisely similar instance (Case 15, with post-mortem examination) was recorded in the 'Reports' for 1857.

CASE 27.—*Acute rheumatic (?) affection of the larger joints; paraplegia of lower extremities; slough over sacrum; recovery.*

Anne E—, æt. 39, was admitted into Guy's Hospital, March 31st, 1857, under the care of my colleagues Dr. Hughes and Dr. Wilks (to whom I am indebted for placing the case at my disposal). Both hands were swollen, stiff, and painful, with an erythematous blush over the back of the right and on the second joint of the thumb of the left. The legs were so far paralysed that she could only very slowly and feebly move them. The muscles were greatly wasted and flabby, but had not lost their excito-contractility by galvanism. Sphincters weak. No swelling of the knees or ankles at this time. Sensation nearly normal, but at times both legs felt numb, and were drawn up involuntarily. Urine acid, high-coloured, and scanty. Tongue covered with a cream-like fur; skin hot, perspiration profuse, with acid smell. Pulse 120; systolic murmur over ventricle. On examining the spine the lower third of the sacrum was found to be bent forward, the result of a fall eleven years before; and near the sacral notch, on the right side, was the cicatrix of a wound which formed at that time. Except this, there was nothing abnormal, nor any pain or tenderness on pressure. The history she gave of her case was, that being a widow, she was necessitated to work laboriously at a mangle. She had for two years, when much exerting herself, felt pain in the back between the shoulders, and a sense of constriction and coldness round the chest. Ten days before coming into the hospital she was seized with pain in the left leg, and had spasmodic contraction of the muscles, with an increase of the pain, and constriction round the chest. She had still power to extend the leg, but could not walk. The day following, the hands, knees, and ankles, were swollen and painful. With these symptoms there was febrile heat and diarrhœa. The sphincter ani was so weak that the fæces ran from her involuntarily. On the third day a slough formed over the sacrum. No important change occurred in her symptoms after her admission. There was great muscular emaciation generally. Involuntary twitchings of the muscles of the arms and legs. Aching, gnawing sensations in both calves. Touching the feet gave rise to formication, and very lively excito-motor movements. For ten days the hands remained red, painful, stiff, and swollen. She complained much of the heat and profuse perspirations, which returned several times in the twenty-four hours. On the 8th of April the urine was ammoniacal, and contained mucus. The hands were still swollen and erythematous; face flushed; pulse 100, full, as in rheumatism; acid smell of perspiration; respiration 28; movements thoracic and abdominal; abdomen soft; pupils large; nights sleepless. Ordered a grain of opium every six hours, with six ounces of wine daily, and a chop. On April 13th the good effects of the opium and support were very apparent. The patient had passed good nights, and was tranquil in the day. Perspiration lessened. Urine retained in the bladder for thirty-six hours was at length passed voluntarily; it was acid, and without mucus. Tongue pale and moist. The slough on the back had deepened. The pupil still continued large. Occasional contraction of the muscles of the legs. No permanent rigidity. Hands remained swollen and stiff, but less red. She was unable to move the shoulders freely. On April 22nd the hands had recovered their normal appearance, and had lost their stiffness. The legs could be moved more freely. The sense of constriction round the chest was gone; pulse 96; skin cool and dry; appetite good; urine normal, but she could not empty the bladder oftener than once in twenty-four hours. From this date she slowly recovered. The opium was continued throughout her convalescence. At the beginning of June the muscles of the lower extremities were galvanized regularly. By the end of the month she was able to stand without help. Her improvement was uninterrupted, and in September she left the hospital quite well.

Remarks.—It is a matter of great clinical interest that lesions of the cord are occasionally attended with an affection of the joints not to be readily distinguished from that which occurs in acute rheumatism. When this happens there may be difficulty in determining the pathology of a case. It may, indeed, be impossible to say whether the symptoms at a certain stage are due to disease of the cord, or to a rheumatic state of the blood. In such instances we have a proof of the near relations of humoralism and solidism; for one observer may maintain that the local lesions have a common origin in the altered state of the blood, whilst another may with equal confidence assert their dependence upon a primary disturbance of the nervous centres. The case here recorded is an example of these difficulties. Fatigue from mechanical labour, acting especially on the lumbar and dorsal portions of the spine in a delicate and anxious subject, appears to have injured the nutrition of the cord. For two years, when much exerting herself, the patient felt pain between the shoulders, and a sense of constriction and coldness round the chest. Paraplegia then suddenly came on, followed by redness, pain, and swelling of the larger joints, as in rheumatism. Together with these symptoms there were others indicating a rheumatic condition—white, furred tongue; flushed face; hot skin; profuse perspirations having an acid smell; systolic murmur over left ventricle, &c. Was there here a rheumatic state of the blood induced by the spinal lesion, or was the nervous derangement the result of a rheumatic state? Notwithstanding the labours of morbid anatomists and chemical pathologists, we are not at present in possession of any certain knowledge of what constitutes the rheumatic condition. My colleague Dr. Addison, from his clinical experience, has long drawn attention to the close connection between spinal lesions and true rheumatism, but has never developed the idea beyond expressing a suspicion of their relation.

At the time this case was under care the treatment was a subject of much observation. The result was very satisfactory. Whatever might have been the state of the cord, it was clearly induced by fatigue, and was soon followed by sloughing of the integuments. It would not, therefore, admit of depletory measures, but, on the contrary, required a nutritious diet and wine. Opium was prescribed apparently with great advantage; it allayed nervous irritability, and gave the patient sleep.

The following case is also illustrative of the relation between spinal injury and rheumatic symptoms. The same plan of treatment as above was equally successful. The therapeutical view of this subject is certainly not without the greatest interest. No doubt the texture of the cord has but feeble reparative powers, notwithstanding it has been shown by experimenters on animals that occasionally, after a transverse section, the parts unite, and the functions are re-established.

Case 28.—*Concussion of the spine; partial paraplegia; redness and swelling of the wrists and ankles as in acute rheumatism; recovery.*

W. T—, æt. 38, on the 22nd January, 1855, inadvertently stepped backwards into a hole, a few feet deep, and received a concussion of the spine. After a few days he became partially paraplegic, with weak sphincters; and at the same time there came on a diffused redness and swelling of the ankles and wrists. The swelling was not from effusion into the joints, but from œdema of the surrounding tissues. The joints were very painful. The redness and swelling were variable in degree. When most marked they presented the usual appearances of rheumatism, or rather of gout, for the erythema was brighter, and the œdema more distinct than in rheumatism. The hands were equally affected with the ankles, though there was no obvious want of muscular power, nor any affection of sensation in the upper extremities. Tongue clean. Pulse 120. No acid perspirations. Urine high-coloured, free from deposits; of normal quantity. The nerves of the surface generally were hyperæsthetic to a slight touch, but deep pressure gave less inconvenience. The treatment consisted of good nourishment, wine and brandy freely administered, and opium to allay pain and overcome sleeplessness. The pulse gradually acquired more power, and sank to 80. The affection of the joints continued in varying degree through March, April, May, and June. From the beginning of April there was an improvement in the power over the legs. The same treatment was continued throughout without the use of mercurials, local depletion, or counter-irritation. In June the patient was able to walk without assistance. During sleep, the hands and feet, wrists and ankles, often became erythematous and swollen. There was occasional formication in the lower extremities. Sleeplessness, from the beginning of the case and throughout, was a troublesome symptom. In July the patient was able to leave the hospital, and to resume to some extent his duties as a medical practitioner. He was under the care of my colleague Mr. Cock.

CASE 29 (Plate III, figs. A, B, C).—*Anæsthesia of left arm without any other symptom. After three years, gradual loss of muscular power in the arm, with wasting of the muscles; subsequently a similar affection of the right arm, but in a less degree. Death from general paraplegia at the end of five years from a fall, by which the anterior columns of the cord were ruptured in the lumbar region. Thickening and adhesions of the meninges, especially in the cervical region of the cord; atrophy of the posterior columns, of the posterior roots of the nerves, and of the grey substance, with a development of fibrous tissue.*

Mary S—, æt. 38, a nurse in Guy's Hospital, complained in 1853 of anæsthesia of the left arm, which had come on gradually for nearly a year. There appeared to be entire loss of feeling below the elbow, but, on testing the sensibility upwards to the shoulder and over the scapula, she gave vague and often contradictory answers, at one time affirming, and at another denying, that she perceived impressions made upon the same points of the skin. This discrepancy was perplexing, and led at the time to the belief that her ailment was either feigned or hysterical. The sensibility at the upper part of the thorax, in the axilla, and at the inner part of the arm, was perfect. The muscles were well nourished, the movements powerful and well directed; but the anæsthesia was so complete that she was unable to hold anything in the hand if her eyes were off it. She often complained of gnawing pains extending down the back, across the shoulders, and into the left shoulder-joint; these pains were increased by the changes of weather. Her symptoms continued unaltered for two years. The following note was made of her case in December, 1855:—"Complete anæsthesia limited to the left arm, no wasting of the muscles, no affection of the leg on the same side, general health in all respects good. Electro-contractility of the muscles of the affected arm good. Electro-sensibility greatly diminished. During the next two years there was gradual loss of power, principally in the left shoulder, but also generally throughout the arm, with marked wasting of the muscles. The right arm became at the same time similarly affected, but in a much less degree. She walked quickly, but with a shuffling gait. The left leg was dragged. She was unable to lift the arms over her head, or to extend them horizontally, but when they hung down she could grasp with tolerable firmness and carry heavy weights. She continued to make frequent complaint of pain in the arms and often down the back, and of a feeling of weight at the epigastrium. Her manner was often excited, her nights restless, and she was subject to attacks of tremulousness and chilliness like ague, with a sense of general numbness. About the middle of December, 1857, she accidentally fell forwards upon the stone steps of the hospital, from stepping upon her dress whilst assisting a patient into a cab. Her left temple was cut, and she was rendered insensible by the fall. On recovering consciousness, a short time afterwards, the legs were found to be quite paralysed, and there was almost entire loss of sensation. The weakness of the arms was greatly increased. There was entire loss of sensation below the elbows, and but feeble traces of sensibility above. The muscles were also much wasted. After the accident, the urine became ammoniacal and contained pus. The skin over the sacrum rapidly sloughed, and she died exhausted at the end of a month.

Post-mortem examination.—General wasting of the muscular system. Lateral ventricles of brain dilated and containing clear fluid. The septum lucidum perforated in many places from atrophy. No disease of the bones or ligaments of the spine. The dura mater of the cord was much thickened, apparently by chronic inflammation. This thickening was most marked at the lower part of the cervical enlargement, and along its posterior surface (Plate III, B, C). In the dorsal region there were plates of true bone, formed by ossific degeneration of the inner layers of the thickened dura mater. One of these plates opposite the third dorsal vertebra was half an inch in length, a third of an inch in width, and a line and a half thick. As these plates were developed by degeneration of the layers of the fibrous membrane, they merely enveloped the cord without producing any pressure upon it. The arachnoid was thickened and opaque, and the two surfaces adherent. In the visceral layer in the lumbar region several cartilaginous (fibrous) plates. These changes were most marked in the neck, but were continuous down to the cauda equina. The texture of the cord itself had undergone important changes, as shown in Plate III. About half an inch below the medulla oblongata, on the left side, there was a cyst occupying the position of the grey matter. Its walls consisted of fibrous tissue and compressed nerve-tissue. There was a similar but smaller cyst on the right side, at a lower level. No more than a trace of it comes into view in the section drawn (fig. A). The cysts contained colourless limpid fluid. At the cervical enlargement, as seen at A, B, the posterior columns and the grey matter were extremely degenerated. They consisted of some remains of the columns, embedded in a stroma of fibrous tissue. The posterior roots of the spinal nerves were included in the degeneration, and the sheaths were thickened in common with the surrounding membranes. The section at B shows this. The lower section at C did not happen to include the nerve-roots, though the same conditions obtained. The anterior columns and portions of the antero-lateral columns were normal, except in the dorsal region, where the anterior columns were ruptured transversely across, apparently at a recent date, and probably by the fall which brought on the fatal symptoms. Viscera of chest healthy. Liver healthy. Acute suppuration of both kidneys; the secreting tissue full of small purulent deposits. Mucous membrane of the pelves dark-coloured and covered with fibrinous exudation. Bladder acutely inflamed; the mucous membrane had sloughed away, scarcely a shred was left on the muscular coat.

Remarks.—The error committed in the early diagnosis of this case was one likely to happen, especially as the patient was a woman. She complained of numbness of the arm. There was nothing visibly wrong with it on the closest examination. The muscles were well developed, the movements were normal, and so were the circulation and temperature. Besides her own account of the numbness there was nothing to indicate disease of the cord or nerves. Her statement, that if she took her eyes off anything held in the hand forthwith she dropped it, was the only circumstance which appeared to have any value as a symptom, and even this was lessened by testing the sensibility. When the patient's head was turned away, and she was unable to see what was done, the point of a needle was passed sharply over different parts of the arm. Below the elbow there was a uniform testimony to the absence of all feeling, but upwards there was every kind of contradiction. When she denied feeling at a part a *minus* sign was put on it with a pen; when she affirmed it a *plus* sign was marked. After mapping out the skin with *plus* and *minus* signs the parts were again tested, and with contradictory results; the *plus* signs fell over the *minus* spots, and the *minus* signs over the spots before marked with *plus* signs—and so on, in the most uncertain way, as often as the trial was repeated. This led to a hasty and false conclusion that the patient was feigning, or that her malady was the vagary of an hysterical state. Further clinical observation in other cases, and the examination of the cord in this, have elucidated what was at its early stage so bewildering. When the sensibility of a part is obscure or doubtful, the testimony of the individual as to impressions made upon it may be also doubtful. The same occurs to us with our healthy sensibilities when, conversely, weak impressions are made upon us. When we look at an object scarcely visible, at one moment it appears, and the next is lost. There is in our minds the same discrepancy as to whether we see it or not, as this patient manifested when asked whether she felt or not. Her contradictions were a proof of the obscurity of her sensations, and her convictions fluctuated between certainty and uncertainty, no doubt because the evidence was to her equivocal.

The lesion began apparently in the membranes, and thence extended to the cord, implicating the sensitive roots of the nerves.

There was no history of any acute invasion, nor did the symptoms at any period indicate acute disease.

The dura mater of the brain occasionally offers a similar form of chronic thickening. Though the morbid change must be referred to inflammatory action, the process must have been most gradual; so gradual, indeed, that the symptoms were only such as were referable to atrophy, although the exudation thickened the membranes and infiltrated the posterior columns. There was no rigidity or other form of spasmodic affection of the muscles, as might have been expected in spinal meningitis.

Whether the exciting cause of the meningeal inflammation was injury, exposure to cold, or a rheumatic condition of the blood, is uncertain. There was no change in the pericardium or valves to corroborate the opinion of its being rheumatic. But, whatever the original cause, its course would be determined by the diathesis of the patient; and hence in the treatment of such a case, we must determine not only the seat and character of the local lesion, but also view it through the peculiarities of the constitution, whether gouty, rheumatic, scrofulous, or syphilitic. Unless we approach accuracy of diagnosis in both these respects, the therapeutics of the case may be no better directed than the efforts of an engineer who should pour medicine down the funnel of his engine because the power fails in the piston.

It is probable that at any early period this case would have been benefited by repeated blisters, and the continued mild use of mercury and iodide of potassium.

The fatal accident was peculiar. The adhesions of the membranes prevented the movements of the cord in the sheath, and exposed it to stretching by any sudden motion of the spine.

The sections of the cord (Plate III) show to what extent disorganisation may take place, and yet the cord serve as a conductor of the voluntary power. The changes at A, B, C, must have been present at the time of the accident, when the patient was able to walk about quickly, and with no more than a shuffling gait and some dragging of the leg.

CASE 30.—*Pain in back and loins for a year; profuse hæmaturia, followed after a month by weakness of the legs, which gradually increased to complete paraplegia; malignant disease of lumbar glands and of the right kidney, extending into the bodies of the vertebræ, and causing sloughing of the cord.*

Mrs. W—, æt. 58, a poor needle-woman, overworked, and but scantily fed, was admitted into Guy's Hospital, December 5th, 1857, under the care of Dr. Wilks, for partial paraplegia of the lower extremities. She had been confined to her bed for eight weeks. There was emaciation of the whole body, but especially of the muscles of the legs, which were loose and flabby. She was just able to stand, but not to walk. The back was straight. No abnormal protrusion of any of the spines of the vertebræ. For a year she had had great pain across the loins and back, with some indefinite tenderness. This was at first supposed to be due to her sedentary habits, and then to rheumatism. A month before her legs began to fail her she had profuse hæmaturia, which was thought to arise from calculus in the kidney. After her admission into the hospital the paraplegia gradually became complete, without any preceding rigidity or involuntary jactitation of the legs. The integuments over the sacrum sloughed, and a similar tendency was manifested over the sides of the knees, from one leg resting on the other. She died exhausted January 20th, 1858.

Post-mortem examination.—The outside of the theca vertebralis was covered with a thin layer of greyish offensive pus. The last dorsal and the three upper lumbar vertebræ were infiltrated with cancer extending from the lumbar glands. The body of the first lumbar vertebra was sloughing. The sloughing process had thence extended to the adjacent portion of the theca vertebralis, and to the body of the cord, which was ash-coloured, and entirely disintegrated, from the eighth lumbar vertebra to the filum terminale. Several broad cartilaginous laminæ in the lumbar arachnoid. No inflammatory exudation within the theca. Above the eighth dorsal vertebra the cord was remarkably pale and flaccid. No discoverable exudation among the tissue. The right kidney was enlarged by cancerous deposit. Left kidney healthy. Uterus and liver healthy. Cancerous tubera on and under the pleura of both lungs, and cancerous deposit in some of the bronchial glands.

CASE 31.—*A wrench of the neck followed after six months by a "stitch" in the neck, supposed to be neuralgic; extensive development of cancer about the upper dorsal vertebræ, throughout the right lung, up the back of the neck under the deep muscles, and inwards between the laminæ of the vertebræ; paralysis of the arm and right leg; softening of the cervical portion of the cord; death sudden.*

Robert P—, æt. 34, a farm labourer, was admitted under my care, August 5th, 1858, for paralysis of both arms and of the right leg. Intelligence perfect. The account he gave of his illness was that six months previously he was taken with a "stitch" in the neck under the right ear. The pain "was so bad, it almost crazed him." After a short time the pain extended to the left side of the neck towards the occiput, and thence downwards between the shoulders into both arms and into the legs. The pain under the left scapula was for a time very distressing. When he had suffered thus for four months, the left arm began to get numb and powerless from the shoulder downwards. He continued able to walk about very well until three weeks before his admission, when the right arm also and the legs began to fail him. The sphincters retained their power for a fortnight longer. On admission, both arms from the shoulders were powerless, but he could move the fingers slightly. Loss of sensation almost complete throughout both arms. Right leg paralysed, left moved with some freedom. Loss of sensation as high as the fourth intercostal space. Left chest uniformly enlarged and universally dull on percussion, including the sternal region. Heart displaced to the right side. Respiration performed entirely by the right lung. Diaphragm and ribs moving freely on this side. Movements of head and neck without pain. Spine straight. No pain in any part. Respiration 44. Pulse 120. The following day, August 6th, at 11 a.m., the breathing became much embarrassed, and he died quite suddenly at 2 p.m. After the post-mortem examination the friends gave an account of his having wrenched his neck about a year before in throwing hay into a loft.

Post-mortem examination.—The left chest equally distended, and the heart displaced to the right side by the development of medullary cancer in the left lung. With the exception of a part of the centre of the lung, the pulmonary tissue was entirely destroyed. The pleura was thickened and cancerous, and firmly adherent to the ribs. In the right lung there was a tumour of the size of an orange, having the usual characters of fungus hæmatodes. The cancerous growth had a firm attachment to the anterior part and sides of the body of the third dorsal vertebra, and extended upwards on both sides of the neck, under the deep muscles, as high as the third cervical, and inwards between the laminæ, so as to come in contact with the theca vertebralis. The theca was thickened, and the trunk of the fourth cervical nerve invaded on the left side. Unfortunately there was no opportunity to dissect the nerves of the brachial plexus, to determine their relations to the disease outside the vertebral canal. The cervical enlargement of the cord was swollen and softened, and granule-cells were scattered through its tissue. This change had apparently advanced into the cord from the right side of the neck. There was no cancerous deposit inside the theca vertebralis. The arachnoid had its normal appearance. It was the substance of the cord only which had begun to suffer from the proximity of the new growth. Head not examined. Viscera of abdomen healthy.

CASE 32.—*Gradual loss of power in right arm, and subsequently in left; after two months and a half, partial paralysis of legs; breathing diaphragmatic; frequent vomiting; pulse quick and feeble. Death by exhaustion, after seven months. Strumous tubercle in the lower half of the cervical enlargement of the cord.*

Elizabeth W—, when eight months old, began gradually to lose the use of the right arm. After a fortnight the left became weak in a similar way. She came under my care as an out-patient at Guy's Hospital, April 13th, 1857, when the paralysis had lasted two months. The wasted arms then hung loose and useless. The head was retracted between the shoulders; the neck stiff. The legs were weak, but could be moved voluntarily. The muscular system generally was wasted, but of the arms most. The skin was constantly warm and freely perspiring. Occasional vomiting. Quick, very feeble pulse. A strumous swelling, the size of a small nut, was noticed in the skin of the right arm. A distinct history of struma on the father's side. The diagnosis was of tubercular deposit in or about the cervical portion of the cord. At the early part of May the right knee became swollen from effusion into the synovial membrane, and from this date both legs became partially paralysed. There were frequent spasmodic contractions in both legs, but most in the right, which was the weaker. In June the breathing was hurried and entirely diaphragmatic. Vomiting frequent. Difficult deglutition. Diarrhœa. During June and July vomiting and diarrhœa continued. There was great heat of skin. Profuse perspirations. Ammoniacal urine. Pulse 140. Respiration 40. The long muscles of back became atrophied. Shoulders drawn up by the elevator muscles of the scapulæ. There still at this time remained traces of voluntary movements in the legs. She died, September 12th, from emaciation and exhaustion.

Post-mortem examination.—Only the cervical portion of the cord was allowed for examination. The surrounding structures were healthy. The cord itself, in the lower half of the cervical enlargement, opposite the origin of the sixth and seventh cervical nerves, appeared to be enlarged.

This enlargement arose from the presence of a strumous tubercle, which at this part had caused complete absorption of the proper tissue of the cord. This formation seemed to have had its origin in the right posterior and postero-lateral columns, thence extending by successive deposits, until the cord was gradually destroyed, only slight traces of the anterior columns remaining where the tubercle was largest. The chief part of the tumour, from the centre outwards, was opaque, yellow, and friable; it consisted of granules, decaying nuclei, cells, and fat. This opaque dead part was surrounded by a transparent thin layer of more recent exudation, consisting of granules, nuclei, and imperfect fibre-cells, with no free oil-globules. Above the tubercle the two layers of arachnoid were firmly adherent, and by contraction had constricted the cord. Just below the tubercle the substance of the cord was so soft that it did not retain its form when unsupported by the membranes.

Remarks.—The gradual onset of the paralysis in this case, and its gradual extension until both arms became paralysed, obviously indicated a progressive organic change in the cord. The nature of this change was also to be plainly inferred from the hereditary tendencies through the father's side, and from the actual presence of a strumous formation in the arm.

That during the earlier stages of its course the disease should have been one of cervical paraplegia, the power over the lower extremities continuing after the arms were paralysed, accords with what has been noticed in other cases; but when instead of central disease the lesion primarily affects the external parts of the cord, at least of the anterior columns, the legs suffer first, and often exclusively if the lesion be moderate.

It is a matter of regret that the state of the sensibility of the legs was not determined. Perhaps, from the age of the child, it could not have been determined.

PLATE II.

FIG. 1.—Transverse section of the spinal cord in the dorsal region (Case 18, p. 216), showing atrophy of the grey substance, and inflammatory degeneration of the columns.

The atrophy did not affect the caudate vesicles. These, by a higher power, were seen to have their normal structure. The white substance was symmetrically degenerated from chronic inflammation. The exudation cells had undergone fatty degeneration, and were incrusted with fat-globules. The capillaries are seen to be similarly incrusted, producing irregular white lines. The symmetry of the lesion was very exact. It included a small portion of the anterior columns on either side of the anterior fissure, the posterior half of the lateral columns, and the centre and posterior portion of the posterior columns. The part of the posterior columns adjacent to the posterior horns of the grey substance was normal. There was no exudation amongst the grey substance. The apparent traces of such, seen in the drawing, are caudate vesicles.

The artist has not strictly drawn the granule-masses according to scale, but he has faithfully rendered the general appearance of the section under a low power.

FIGS. 2 and 3.—Transverse sections of the cord (Case 19, p. 219) showing degeneration of the posterior columns from chronic inflammation.

The anterior and antero-lateral columns, and the grey substance, were normal. The upper section (fig. 2) is from the upper cervical region; the lower (fig. 3) from the lower dorsal region. The granular appearance was due to fatty degeneration of the inflammatory exudation. Though the artist has exaggerated the relative size of the granules to the columns for distinctness' sake, he has strictly maintained their relative distribution. The lesion was remarkable from its being so entirely limited to the posterior columns, though it affected them throughout their own length.

PLATE II.

Fig. I.

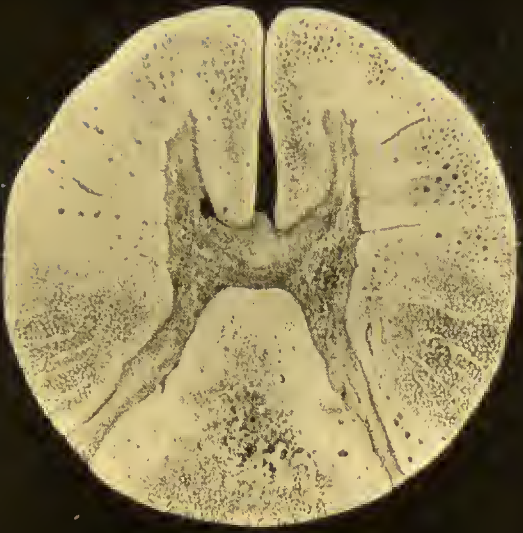


Fig. II.

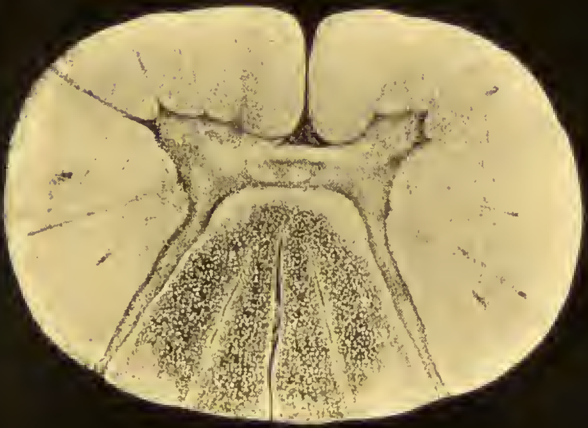


Fig. III.

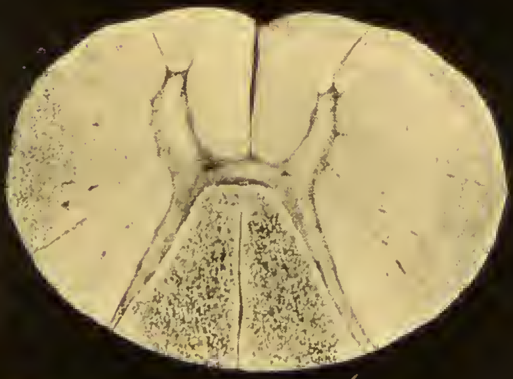


Fig. I, Case 18, p. 216.
Figs. II, III, Case 19, p. 219.

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PLATE III.

Transverse sections of the cord and its membranes, Case 29, p. 236.

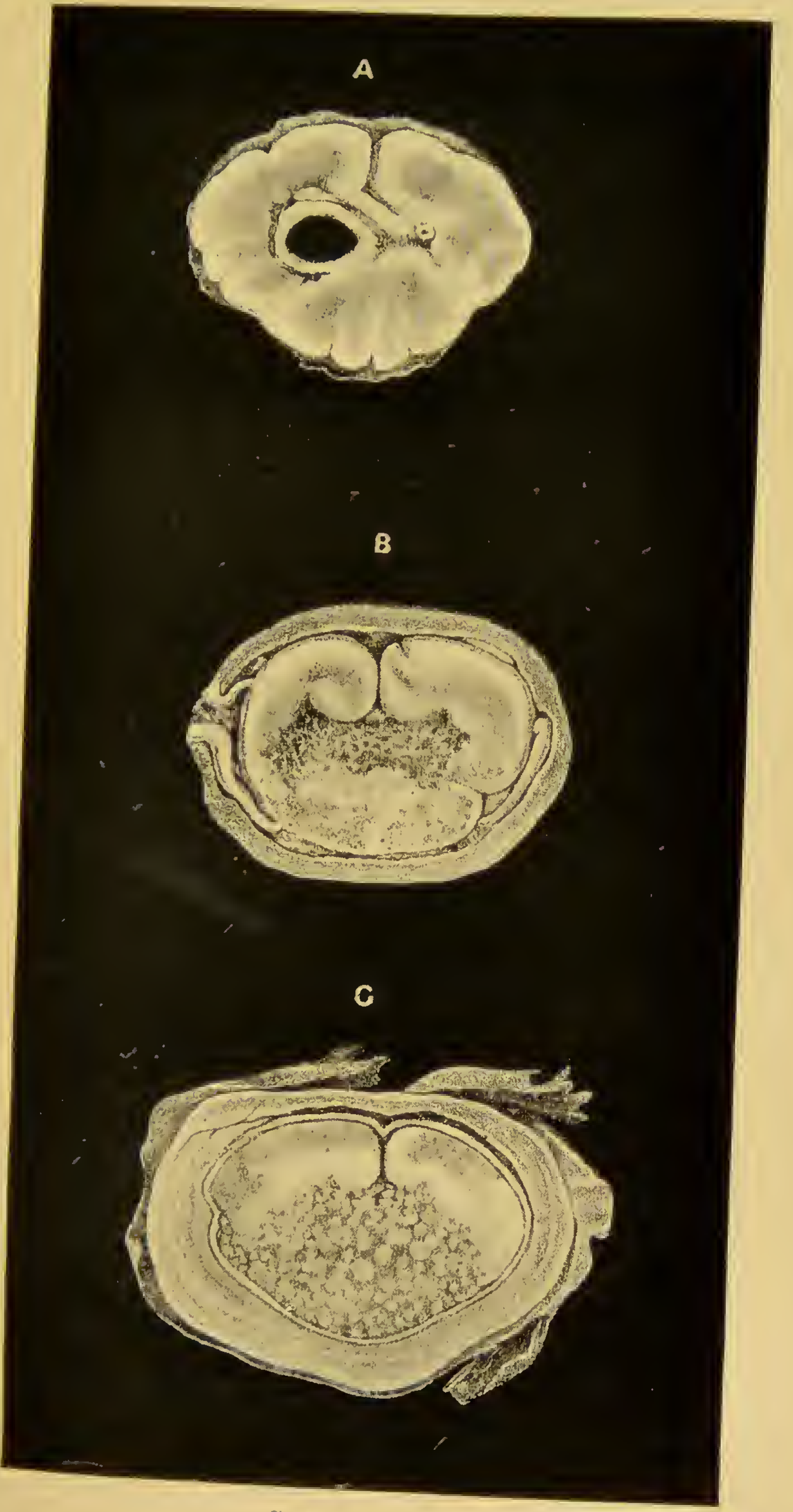
A. Section through upper part of the cervical region. The left side of the cord was here distorted by the development of a cyst in the grey substance. A smaller cyst of the same kind existed in the grey substance on the right side, but at a lower level, so that only a trace of it is visible in this section. These cysts had distinct walls of fibrous tissue and condensed nerve-substance. They contained clear colourless fluid.

B. Section through upper part of the cervical enlargement, showing great thickening of the membranes, and degeneration of the posterior columns and grey substance, including also the posterior roots of the nerves, with the development of common white fibrous tissue in place of the normal structures.

C. Section about the middle of the cervical enlargement. The membranes, and especially the dura mater, extremely thickened. This change was greatest on the posterior surface of the cord, where the membranes were adherent together. The posterior columns, the grey substance, and the posterior roots much degenerated. Some of the normal structure of the posterior columns is seen lying embedded in a stroma of fibrous tissue.

These changes were due to chronic inflammation, apparently advancing from the membranes into the substance of the cord.

PLATE III.



Case 29, p. 236.

Adlard and Son, Imp.

CASES OF PARAPLEGIA

ASSOCIATED WITH

GONORRHŒA AND STRICTURE OF THE URETHRA.[1]

In the year 1833 Mr. Stanley read before the Society some cases of paraplegia arising from primary disease of the urinary organs.

According to the views expressed by him, disease of the kidneys may produce a morbid impression upon the cord through sentient nerves, which, being reflected outwards to the extremities, may occasion an impairment of both motion and sensation, and paraplegia result without organic lesion of the cord itself.

It would require a more minute examination of the cord than was made in the cases given in the paper referred to before the important negative assumed in their explanation could be regarded as established, since it is known that the structure of the cord may be extensively disorganised where an experienced observer, without the aid of the microscope, may fail to discover the traces of disease. In proof of this I may quote the following case, which was under the care of my friend and colleague, Mr. Hilton, who has kindly placed it at my disposal.

Paraplegia following gonorrhœa and syphilis; inflammation of the substance of the cord; no traces of lesion discoverable without the microscope.

James L—, æt. 20, admitted into Guy's Hospital March 14th, 1855; a gentleman's servant, unmarried. Always had good health until he contracted gonorrhœa and had a chancre eight months ago. He was under treatment for three months. After the chancre had healed he again became infected, and ulceration followed at the seat of the old cicatrix. For this he was again under treatment until the beginning of the year 1855. At that time (January 18th), having occasion to go from home, he slept, as he thinks, in a damp bed, and three days afterwards began to have pains and weakness in the legs and about the neck and occiput.

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[1] Reprinted from the 'Medico-Chirurgical Transactions,' 1856, p. 195.

On the 26th he had a rigor, and the weakness of the legs was rather suddenly increased, with loss of sensation above the ankles and formication in the feet. Incontinence of urine came on at the same time.

On the 28th he managed to get downstairs with the help of his mother and the use of a crutch, but at night he had lost all power in the legs, and was carried to bed. During the next fortnight the loss of sensation gradually extended upwards to a line corresponding to the distribution of the ninth dorsal nerve. The sphincters were paralysed. The susceptibility to the excito-motor movements continued to increase, and the cord at length became so irritable as to occasion the patient great distress; the least agitation or the slightest touch bringing on violent spasmodic contractions of the legs, though the irritation was quite unfelt. There was a painful sense of constriction across the chest. Bedsores formed and rapidly extended.

There was no important change in his symptoms until April 18th. The urine was ammoniacal and continually dribbled from him, excoriating the scrotum and inner parts of the thighs. The bedsores sloughed. There were frequent involuntary spasms of both legs, but especially of the left. At this date he began to have cough, headache, and more frequent rigors. Tongue became furred. Pulse accelerated.

He died rather suddenly, May 16th (four months from the beginning of his symptoms), having during the last month become much exhausted from frequent rigors and hectic.

On a *sectio cadaveris* the vertebral canal was healthy. On opening the dura mater the two layers of arachnoid were found united, as usual, on the posterior surface of the cord by delicate adhesion. There were some osseous plates on the visceral layer of the membrane. No traces of vascular injection or of inflammatory exudation. *The cord had the normal size and appearance, and neither to the touch nor on section presented any obvious softening.* With a lens of an inch focus the surface of the columns at and below the origin of the sixth nerve had a mottled appearance, some portions being opaque and yellowish; and a more minute microscopical examination discovered extensive disorganisation of the nervous structure, the focus of the morbid change being at the middle of the dorsal region and principally in the anterior columns. The fibrous structure was loose, and amongst it, and apparently resulting from its disorganisation, were numerous oily granules, together with a great number of the characteristic mulberry masses (granule-cells). Sections of the cord at the lower part of the dorsal and in the cervical region gave the same results, but in a less degree.

This proves that we ought to look with great mistrust upon the evidence which the unassisted eye supplies in the examination of nervous structures, where but slight lesions produce such decided and striking symptoms.

The following cases seem to show that, instead of regarding the nerves as the channels through which the cord is secondarily affected in disease of the urinary organs, we ought rather to look to the *veins* or *the blood itself* as the means by which the lesion is propagated, and, instead of attributing the paraplegia to functional depression of the nervous energies, to refer it to inflammatory changes.

In the following case this pathological relation certainly existed. For the particulars of it I am indebted to my friend Dr. Habershon.

Paraplegia; acute spinal arachnitis and softening of the cord following retention of urine from stricture.

William W—, æt. 29, a cabman, admitted into Guy's Hospital on Sunday morning, September 19th, 1847, for retention of urine and stricture, to which he had been subject for several years. After a warm bath, and with some difficulty, the smallest catheter was passed and the urine drawn off. On the following day he had again difficulty in emptying the bladder, and twenty leeches were applied to the perinæum. From this date until the 28th the stricture was dilated daily, and he was going on favorably, being a considerable part of the day up and about the ward, apparently in his usual health. On the 28th he complained of a fixed and constant pain near the angle of the tenth rib on the right side, for which a blister was applied, with relief. Three days after (October 1st) he was free from pain, but feverish. He dressed himself as usual and sat by the fire; but, on attempting to return to his bed in the afternoon, he suddenly found his legs weak and numb. Pulse 120. Tongue thickly furred. He was freely purged without benefit. On the 3rd the loss of sensation and motion was complete in both legs, and sensation was imperfect on the surface of the abdomen as high as the umbilicus. He had no pain in the spine, nor any convulsive movements of the legs. The bladder was emptied morning and evening by the catheter. In the intervals it dribbled away, highly ammoniacal and purulent. Mr. Key, under whose care the patient had been admitted, saw him on the 5th, and considered the paralysis to depend upon thickening of the posterior common ligament.

He gradually became more prostrate. A large slough formed over the sacrum. The evacuations passed involuntarily. He expired on the 27th, one month from the commencement of the spinal symptoms. There was no affection of the brain throughout.

Sectio cadaveris.—Head not examined. On removing the cord with its membranes from the canal a small quantity of pus was found lying on the outside of the sheath, opposite the bodies of the sixth, seventh, eighth, and ninth dorsal vertebræ, and one of the vertebral veins in the lumbar region was full of well-formed pus. The spinal fluid was densely coagulable. The arachnoid was thickened and presented traces of recent inflammatory exudation. The dorsal portion of the cord was very distinctly and generally softened. Lungs healthy. Pericardium contained a small quantity of fluid, with a patch of fibrin upon the ventricle. Liver healthy. Kidneys large and congested, with spots of ecchymosis. Secreting structure coarse and soft. Pelves dilated; their mucous membrane and that of the ureters and bladder injected and covered with purulent exudation. An old stricture existed at the commencement of the membranous portion of the urethra, and several false passages, one opening into an abscess behind the bladder, and two returning into the bladder. The vesical veins in the neighbourhood of the pelvic abscess were thickened and partially obstructed by recent lymph. No traces of peritonitis. Intestines healthy.

Here phlebitis was no doubt caused by the catheterism, but in the following cases gonorrhœa probably brought on paraplegia as one of its proper sequelæ, after the manner in which its other secondary affections, as swelling of the joints, are produced. Whether this be through a purulent infection of the blood, or through some more specific taint, is yet unknown.

Paraplegia, with softening of the cord, following gonorrhœa and chronic gleet.[1]

Henry F—, æt. 21, a pale and delicate man, a shoemaker. Habits irregular. Has had gonorrhœa many times, and is subject to a permanent gleet, increased when he indulges in drink. His general health has been good; and he was, so far as he knows, quite well on Tuesday morning, March 1st, 1853. In the afternoon of that day he began to have pain between the shoulders, and a diarrhœa came on, to which he had been frequently subject. This continued during the night, with increased pain in the back and spasmodic tremblings in the legs. Towards morning the legs became weak and numb, and he was unable to void his urine. His friends, for his relief, applied hot fomentations to the feet, legs, and pubes, which produced extensive vesication. He was brought to Guy's Hospital, March 4th, and admitted under the care of Mr. Bransby Cooper, with the following symptoms:—complete loss of motion below the sixth dorsal vertebra; the muscles of the seventh intercostal space do not act in respiration; sensation perfect above the line indicated, but on the abdomen pinching or pricking the skin gives no pain, and only the faintest sensation; in the legs there is complete anæsthesia; bladder distended, with dribbling of urine; great exhaustion; pulse 110, weak; respiration tranquil, 24;

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[1] Cf. a lecture on Urinary Paraplegias in the New Sydenham Soc. vol., 1881, 'On Diseases of the Nervous System,' by T. M. Charcot, p. 252, in which this case is quoted.—Ed.

febrile heat; tongue injected; complains much of thirst. Spine quite straight. A slight degree of tenderness and some sense of stiffness between the shoulders. At the epigastrium and about the penis, thighs, and ankles the integuments are vesicated and the skin is in parts sloughing, from the hot fomentations which have been applied; and last night, in addition, several bullæ formed spontaneously on the left ankle and on the soles of the feet. No bedsore. Fæces healthy, passed involuntarily. Urine drawn off by the catheter, ammoniacal, and containing mucus and pus, with traces of blood.

He died exhausted from irritative fever and sloughing, March 15th, a fortnight from the beginning of the paraplegic symptoms.

Sectio cadaveris.—Several superficial sloughs over the legs and abdomen. Large sloughing bedsore over sacrum. Bullæ on the soles of the feet. Bones and ligaments of the spine healthy. The cord was generally softened as high as the middle of the dorsal region, at which point the nervous substance was broken up by the gentlest stream of water falling on it. The grey and white portions appeared to be equally affected. Amongst the softened nerve tissue granule-cells were abundant. There was no point of suppuration, nor any trace of old disease in the cord. The membranes were apparently healthy; liver healthy; kidneys of a dark colour, from venous congestion; the mucous membranes of the pelves slightly ecchymosed; bladder thickened, the lining membrane covered by recent diphtheritic exudation. Between the bladder and rectum there was an irregular abscess, with sloughing walls, communicating with the bladder by a large perforation of its coats. Near the bulb was a more recent abscess, filled with healthy pus. The lungs collapsed freely on opening the chest, and were free from disease. Heart healthy.

Paraplegia occurring with gonorrhœa; recovery.

Alfred L—, æt. 28, a thin, pale young man, below the middle stature, employed in a ready-made shoe warehouse. His habits are rather irregular, but yet he has had no illness until the present attack. On the 17th of June, 1855, he took a gonorrhœa, attended with the usual symptoms, until four days ago, July 1st, when he began to feel pain between the shoulders, which he attributed to cold, though he knew of no exposure. The following day the pain had increased, and extended to the loins, but was not such as to prevent his going on a Sunday excursion to Brighton. On reaching town in the evening he walked to his home, the distance of a mile and a half, without feeling any weakness in his legs. The next day (July 3rd) he was at his work, but felt very unwell, and his legs weak; he had some difficulty in emptying the bladder. In the evening he applied a mustard poultice to the loins, and passed a quiet night. July 4th he was unable to leave his bed, and sent for my friend Mr. Roper on account of retention of urine. During the day the legs became weaker, and in the evening he again required the use of the catheter, and his evacuations passed from him involuntarily. July 5th he was admitted into Guy's Hospital, under my care. He could move the legs only very feebly; numbness along the outer part of the thighs as far as the knees; involuntary muscular twitchings in both legs; abdomen flaccid, a sense of constriction around the lower part; inability to empty the bladder; constipation; no tenderness in the spine; no affection of the joints. Tongue moist, slightly furred; skin cool; pulse 76. Moderate gonorrhœal discharge.

By cupping, laxatives, and counter-irritation, the paralytic symptoms slowly disappeared, and on the 15th he was able to stand without help, though not to walk. A third blister was applied to the loins, and medicine continued, until he became impatient to return home. When he left the hospital he had but slight weakness in the legs. The gonorrhœal discharge had gradually ceased.

He continued to attend as an out-patient for a month, and was then apparently well.

I saw nothing more of him until July, 1856, when he again applied on account of a slight return of his spinal symptoms, of which he maintained he had no traces until the occurrence of the gonorrhœa.

By a return to his former treatment he again improved, and is now well.

That the occurrence of paraplegia with gonorrhœa is due to an inflammatory affection of the cord is further made probable by one at least of Mr. Stanley's own cases, which I may perhaps be allowed to quote in this place:

"A man, æt. 30, was admitted three weeks previously on account of gonorrhœa, with phimosis, which was in progress towards cure; the inflammation in the urethra had subsided, but the discharge continued. Whilst in this state as far as the local disease was concerned, and without any particular derangement of the general health, he was suddenly seized with paraplegia, which extended as high as the umbilicus. In the limbs the loss of motion was complete, and the loss of sensation nearly so; the functions of the brain were unaffected. On being questioned, he stated that he had been suffering for a day or two from pain in the loins. The pulse was 85, and rather full. He was cupped in the loins, and free action of the bowels obtained by purgatives, but with no benefit. The urine flowed involuntarily, and in considerable quantity. As, however, it was thought the bladder was distended, a catheter was introduced, and three pints evacuated. In sixteen hours from the attack of paraplegia the man suddenly fell back in his bed and died. The spinal cord was first carefully examined. *There was found some turgescence of the vessels, both in the membranes and substance of its lumbar portion*, and a few drachms of transparent fluid in the theca; 'but,' says Mr. Stanley, 'neither the turgescence of vessels nor effusion of fluid was sufficient to explain the paraplegia by pressure on the cord.' The liver was enlarged and indurated. The other abdominal viscera, with the exception of the kidneys, were sound, and with no unusual turgescence of the vessels. Both kidneys were of so dark a colour as to be almost black; they were remarkably flaccid, and, on sections being made of them, were found to be in every part gorged with blood. The mucous membrane lining the infundibula and pelves was dark-coloured, from the turgescence of the vessels. The coats of the ureters and the mucous membrane lining the bladder were also very much more loaded with vessels than is usual. In the bladder was about a pint of urine. Some fluid was found between the membranes of the brain and in its ventricles."

The objection made by Mr. Stanley that the turgescence of the vessels and the effusion of fluid were insufficient to produce paralysis by pressure is no doubt valid, but it equally applies to a large proportion of the cases of paraplegia from inflammation of the structure of the cord. It is not often the amount of exudation which by its mechanical action determines the paralysis, but rather the coincident changes in the nervous tissue from defective nutrition and softening. The small amount of exudation, and the apparently slight changes of structure which accompany the inflammatory lesions of the cord, is one of the most remarkable points in their history; and here it is, as I wish to prove, that the microscope has so much aided our investigations. With the knowledge we have so obtained, the *injection of vessels in the structure of the cord* must be considered an important indication of organic lesion, and can leave us in but little doubt that the paralysis was the result of disease of the cord, rather than of any simply morbid impression made upon it through incident nerves.

ON

PARALYSIS OF THE LOWER EXTREMITIES,

CONSEQUENT UPON

DISEASE OF THE BLADDER AND KIDNEYS.

(URINARY PARAPLEGIA.)[1]

It is admitted as an established fact in medicine that paralysis of the lower extremities is occasionally caused by diseases of the urinary organs; but observers are not agreed upon the state of the spinal cord in these cases, nor upon the character of the morbid processes by which the cord is affected.

The diagnosis of urinary paraplegia is not free from some fallacies. These have not been sufficiently recognised by writers, or rather they have been passed over altogether. The evil of this course has been great, since it has vitiated the general conclusions arrived at. The cases recorded need a winnowing criticism. If they had received it, the clinical history of urinary paraplegia would have had a more secure basis, and there would have been no room for this communication.

Urinary paraplegia, as the name implies, is paralysis set up by disease beginning in the urinary organs.

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[1] Reprinted from the 'Guy's Hospital Reports,' 1861, p. 313.

The first difficulty in the diagnosis is to distinguish the true order of the morbid processes from that which they superficially present to our notice.

We require but little clinical experience to inform us that symptoms and lesions may become apparent to us in an order very different from their true one; and that we cannot, without collateral proof, determine what the true order is. So fallacious is often the apparent relation of phenomena, that it may tax the most matured experience to avoid error.

We might refer, in illustration of this, to the history of cerebral disease. It is notorious that organic affections of the brain may for months or years lie latent, and bewilder us with peripheral symptoms, and particularly with every variety of gastric disturbance, until some definite and often sudden warning from the brain unmasks the truth to us, and proves how erroneous our thoughts have previously been.

It is not always easy to determine at the outset whether symptoms have a peripheral or central origin. In paraplegia associated with disease of the urinary organs, the difficulty is perhaps greater than elsewhere; yet it has been tacitly assumed that, if cystitis or other lesion of the bladder precede paralysis of the lower extremities, we are to infer that the paralysis has had a peripheral origin, and has been occasioned by the peripheral lesion which first prominently arrested our attention. This, at least, is the conclusion which must be drawn from such a statement as the following:

"To give at once," says Dr. Brown-Séquard, "an idea of the striking differences between the kind of paraplegia which deserves the name of reflex and one of the forms of paraplegia of centric origin, we will condense in the following table[1] the principal features of two of the most characterised varieties of reflex and centric paralysis of the lower limbs—*i. e.* of paraplegia due to a disease of the urinary organs, and paraplegia due to myelitis."

He goes on in the table to say that urinary paraplegia is preceded by an affection of the bladder, the kidneys, or the prostate, whilst in paraplegia from myelitis there is no disease of the urinary organs, except as a consequence of the paralysis.

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[1] 'Lancet,' April, 1860.

Taken thus generally, nothing is more untenable than this statement. We might equally maintain that gastric affections which lead off the course of symptoms in diseases of the brain are the cause of the cerebral disease, as that the urinary affection is the cause of the paraplegia which it precedes. Disease of the bladder, prostate, or kidneys may be the cause of paraplegia; but it is equally true that affections of the bladder, kidneys, or prostate may be the prelude of a paraplegia from myelitis. Therefore, so far as precedence of symptoms goes, nothing can be inferred from that fact alone. There is, indeed, one form of paraplegia, which usually begins with symptoms of catarrhus vesicæ, namely, that form which depends upon cerebro-spinal exhaustion from mental overwork. We see the distant threatenings of it, and happily for the most part only distant, in the faintly alkaline urine, turbid from the phosphate of lime, and attended with lumbar malaise, which comes before us almost every day in this overworked metropolis.

As an example of this form of paraplegia the following case may be here recorded:—A gentleman æt. 54, of anxious temperament, and engaged as manager of a public company, consults his medical attendant for a deposit of mucus in the urine. There appear to be no other symptoms to arrest attention. The gait is firm, and a day's shooting can be taken over the country without unusual fatigue. Nothing is discovered at this stage of the case to account for the cystitis, but various theories are suggested. Amongst others, it is attributed to a gouty affection of the bladder, or to a calculus in the kidney. These explanations seem the more probable from the occurrence of flying pains in the lower extremities. Months go on, the vesical discharge continues, but there is no suspicion of its being due to a morbid state of the cord. The legs show no distinct weakness. The stomach becomes irritable. With this new development of peripheral symptoms, there is still no suspicion of any affection of the cord. Two years from the commencement of the disease of the bladder, and some months after the irritability of the stomach began, the lower extremities became weak; and now for the first time it was suspected that a failing state of the nervous centres had given rise, from the beginning, to the vesical and gastric symptoms, and were themselves indeed the first evidence of such a failure. Other symptoms, previously unnoticed, could now be observed; the contracted pupils, the occasional vertigo and headache, the inability to apply the mind with vigour, and the wandering rheumatic (?) pains. Arrived at this stage, the case exhibited an instance of paraplegia, in which the earliest effects of the paralysis were evinced, through the sympathetic, upon the bladder and stomach, as a flow of tears follows an emotional affection of the brain.

It is no new fact in medicine that cerebral exhaustion may impair the functions of the cord (especially of its lower segments), and give rise to precisely those symptoms which have been set down as pathognomonic of urinary paraplegia. Illustrations might be multiplied in support of this assertion; but, as common experience will supply such, they need not be introduced here. I cannot, however, refrain from referring to one other case, in which severe dyspepsia preceded for a long time the symptoms of paraplegia from myelitis. It occurred in a man æt. 58, whose nervous power had been depressed by much mental distress. Together with the gastric pain and irritability, there was in the urine a large deposit of uric acid. Dyspepsia and these uric acid deposits continued from autumn to the spring of the following year before any weakness of the legs became evident. There was not then complete paralysis. The legs were only very weak, and the degree of weakness was variable. For many months the progress of the case was marked by similar fluctuations, until at length the paraplegia was complete. Sensation was never diminished; but, on the contrary, there was in the early stage of the case an over-sensibility of the surface. On a post-mortem examination the entire substance of the cord, in the middle of the dorsal region, was softened almost to diffluence.

A recognition of this common source of error, in respect to the true order of the morbid processes, lies at the threshold of the diagnosis of urinary paraplegia. There is, perhaps, no fact to be more insisted upon than the dependence of the normal functions of the sympathetic upon the integrity of the spinal system. As a result of this dependence we learn that dyspepsia, vomiting, constipation, colic, vesical catarrh, prostatic irritation, pains in the joints, and many other peripheral disturbances, may seem to precede the central malady, and to be the cause of it, when in truth they are its effects.

It would not be difficult to point out examples of this clinical fallacy of *hysteron proteron* in the writings of even great authorities. In reference to the present subject, we might instance the cases of paraplegia said to be due to enteritis; nor should we be deterred from quoting them as examples of this error, though supported by such authority, since science recognises no authority but that which is based upon evidence. Striking as such cases at first perusal seem, they fade away under inquiry, and take no rank as established truths. The existence of enteritis, assumed as the active cause in these cases, is itself more than doubtful, and the paraplegia said to have ensued from it is supported by no better evidence.

There is a second source of error not alluded to by writers on urinary paraplegia. As if anxious to prove the existence of the disease more by bulk of evidence than by the character of it, they accept the mere muscular weakness which is often associated with prostatic and vesical disease for partial paraplegia. A perusal of the treatise of Leroy d'Etiolles, jun.,[1] to which such frequent reference is made, will bear out this statement, if we may accept his words at their strict value. Whilst at the heading of many of his cases there is the word "paraplegia," in the subsequent details of them we find no further evidence of paralysis than may be gathered from such expressions as the following:— "faiblesse dans les jambes;" "faiblesse des membres, telle qu'elle ne permet plus au malade de se tenir debout sans appui;" "qu'il ne pouvait se soutenir sur ses jambes;" "le malade se sent faible sur les jambes, et après la moindre fatigue il éprouve des tremblements, il ne peut marcher sans le secours d'un bâton;" "affaiblissement des membres inférieurs;" "des tremblements," &c. These expressions, though intended to convey evidence of paralysis, are not in themselves any proof of its existence. Nor does the perusal of many of these cases lead us to the conclusion that there was in reality any paraplegia at all.

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[1] 'Des Paralysies des Membres inférieurs,' &c., Paris, 1856.

If we bear in mind how much the activity of the lower limbs depends upon an unfettered action of the abdominal and lumbar muscles, and of the psoæ, iliaci and glutæi, we cannot but recognise a fertile source of "pseudo-paraplegia" in that impairment of muscular activity which necessarily attends the malaise of chronic pelvic affections, whether vesical or uterine.

A perusal of the cases which have been recorded or quoted by authors, to establish the frequency of paraplegia from pelvic disease, will show that the error here pointed out has not been unfrequent. And since in these instances of what we here term "pseudo-paraplegia" the cord is not implicated, neither the recovery of the patient nor the absence of lesion after death can be brought in support of any theory of paraplegia properly so called.

To those who are unacquainted with the difficulties of the subject, the existence of paraplegia in any case may seem a matter too obvious for discussion. There is, however, often difficulty, or at least necessity for care, in deciding whether there be paralysis or not.

To illustrate this point I may record the following case, in which there was such universal muscular atrophy as to render the patient incapable of moving the lower extremities with any power or freedom, or of carrying the hands to the head. Although there was no paralysis, still the impression that it was a paralytic affection was at first so obviously suggested by the wasted and powerless limbs, that it needed a careful consideration of the facts to avoid falling into error.

For the details I am indebted to Dr. Francis Hutchinson, of Blackfriars, with whom I saw the case, and who kindly obtained for me a post-mortem examination.

Universal muscular atrophy to an extreme extent (pseudo-paraplegia); anæmia.

Mrs. C—, descended of a healthy family, was in good health until the age of thirty-two, when she lost her only child. From this time she became intemperate, and continued her intemperate habits through many years. At the age of forty-one her muscular system rapidly emaciated. All useful power of the upper and lower extremities was lost; the hands dropped at the wrists, and the feet could not be raised at the ankles. Except transient formication, returning at short intervals for a day or two, there was no affection of sensation, nor was there any pain. Under the use of tonics and galvanism there appeared to be some slight improvement. I saw her three months before her death; she was extremely sallow. The whole muscular system was remarkably wasted; she could not stand, nor even carry her food to her mouth without supporting one arm with the other. Sphincters good; deglutition good. More or less general tenderness of the surface, but otherwise no hyperæsthesia, the tenderness being attributable to her attenuation. The special senses and the intellect unaffected; speech good.

During the two months preceding her death the hands recovered much of their power, so far that she could grasp a cup and raise it to her lips, but she could not pick up a pin or thread a needle. She was cheerful; memory slightly impaired. There was at times that hallucination which occurs in the general paralysis of the insane. She often said she could walk about, and seemed to be unaware of her helpless state. At other times there was no intellectual disturbance, and that which is noted was difficult to detect. The sphincters were throughout unaffected, and the expression of the face betrayed no cerebral disturbance. Five days before her death febrile symptoms set in, with slight wandering delirium.

Although the first aspect of this case gave the impression that it was one of paralysis from disease of the cord, there was in reality no paralysis at all. The dropped and extended feet and partially dropped wrists, and the thumb gently inclined to the palm, were owing to the muscles being too weak in their nutrition to counteract the mere force of gravitation. The case was strictly one of debility, and not of paralysis, if we may distinguish cases of diminution of voluntary power from simple insufficiency of muscular nutrition from those of actual lesion of the nervous or muscular apparatus. The following were the post-mortem appearances:

Post-mortem examination.—Cord small, especially at cervical and lumbar enlargements; texture firm; no abnormal elements discovered in the tissue by the microscope. Membranes of cord healthy; anterior roots of nerves normal. The anterior and posterior columns maintained their due proportion to the other parts of the cord; head small; calvarium very thick, especially at the frontal protuberances. Much clear subarachnoid effusion, supplemental to the wasted brain; lateral ventricles rather large; septum lucidum very thin. The cerebral substance had throughout the normal firmness and appearance on section. The heart was small and flaccid, its tissue soft and easily lacerable, the right ventricle much loaded with fat; the muscular fibre had not undergone fatty degeneration. The liver large and pale, more or less universally cirrhosed; kidneys normal. The voluntary muscles of the extremities and the intercostals were pale and wasted; the fibrillæ not fatty.

Urinary paraplegia occurs but very rarely in women.

It occurs more commonly in men, and generally as a consequence of long-continued chronic disease in the urethra and neck of the bladder.

Children, though liable to calculus and the irritation consequent upon it, do not appear to suffer from urinary paraplegia.

It is important to note that, except in acute gonorrhœa, urinary paraplegia does not occur until after the chronic affection of the urethra and bladder giving rise to it has lasted some time—*often many years.* The history commonly runs thus :—" Stricture many years ;—frequent catheterism ;—at length numbness and tingling in legs, and imperfect paraplegia." Or, " Stricture ;—catheterism ;—intermittent febrile symptoms ;—and, subsequently (at an indefinite period) paraplegia."

In some cases ten or more years have intervened from the commencement of the pelvic affection to the onset of paralytic symptoms in the legs.

Urinary paraplegia is not known to follow the irritation produced by the passage of urinary calculi.

Neither does it follow the casual introduction of a catheter or sound in exploring the bladder for a calculus.

It rarely follows cases of primary pyelitis, whether produced by renal calculi or by strumous disease.

In unequivocal cases, by which I mean those in which the affection of the cord is directly referable to the pelvic disease, there is commonly pain in the back preceding the paralysis. But the pain in the loins may be absent even though there be acute inflammation about the cord. In one case[1] the spinal veins were in a state of suppuration, but there was no pain in the back. It may be asserted, not of urinary paraplegia only, but also of inflammation of the membranes and substance of the cord occurring under other circumstances, that pain, in its presence and degree, is very variable; and that it is more dependent upon the implication of the nerves in the canal, and of the muscles, bones, and ligaments which surround the cord, than upon the state of the cord itself or its membranes.

In many cases irritative fever precedes the development of urinary paraplegia, the febrile symptoms being often directly referable to suppuration in or about the prostate or neck of the bladder.

Commonly, only the lower extremities are affected. This, however, is not constant; for the disease may extend up to an indefinite height in the cord. We cannot give a better instance of this than the case of Professor Sanson, so often quoted. This celebrated French surgeon was for many years liable to prostatic irritation, for which sounds were employed and cauterisation. An ammoniaco-magnesian calculus formed in the bladder and was broken up. A year before his death (after years of suffering) he began to complain of numbness and painful formication in the gluteal region. In a short time there was complete loss of sensation in the lower extremities, and subsequently as high as the mammæ. Firm pressure or percussion over the upper dorsal vertebræ gave pain. The lower extremities became permanently flexed and rigid, and violent convulsions came on in them, sometimes spontaneous, sometimes excited by tickling or pinching the feet, although the patient was not aware of the stimulus. The symptoms underwent no amelioration until his death.[2]

This case was undoubtedly one of urinary paraplegia, yet its details do not at all tally with the characters which

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[1] 'Medico-Chirurgical Transactions,' vol. xxxix.

[2] We reproduce this case here in its general details, not only because it exhibits this extension of the disease upwards in the cord, but because it shows the insufficiency of the theory of vaso-reflex paralysis of which it has been adduced as an illustration.

are given of urinary paraplegia as a reflex disease. Far from there being any correspondence between the theory and the facts, there is a difference in almost every particular. But, to return to the question of the limitation of paralysis to the lower limbs, it may be asserted that no deduction can be drawn from this as to the cause of the paralysis. Such limitation can of itself mean no more than that the lesion, whatever it may be, affects only or chiefly the lumbar segments of the cord. It is certainly no test of the nature of the lesion, and no peculiarity of urinary paraplegia. The same limitation occurs not unfrequently as the effect of primary myelitis, and also of that condition of the cord which we recognise, but which we can no further explain than by saying it arises from central exhaustion (asthenia).

Such cases have already been alluded to, nor would it be necessary again to refer to them had not a case of this kind, recorded in the 'Guy's Hospital Reports,'[1] been quoted by Dr. Brown-Séquard in support of his theory of vaso-reflex paralysis, as one of urinary paraplegia. It is remarkable that it should have been so quoted, since there was no disease of the urinary organs preceding the paralysis, and none until the paralysis made it necessary to use catheters.

Although we should not be disposed to lay any great weight upon the fact, since we do not admit the validity of the distinctions which have been given, it may be further remarked that a comparison of this case with the characteristics of urinary paraplegia, set down according to the vaso-reflex theory, shows that it could not, even theoretically, have belonged to that class. Its symptoms contradict most of those ascribed to urinary paraplegia. To make this plain we subjoin the case itself, and a table of the symptoms in urinary paraplegia.[2] The numbers placed between brackets correspond to those in the table.

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[1] 'Guy's Hospital Reports,' 1858.

[2] 'Lancet,' April 21st, 1860.

Complete paraplegia without loss of sensation; onset of symptoms sudden; death after fourteen days, from acute peritonitis set up by inflammation of the bladder; no discoverable change in the structure of the cord beyond slight softening of the texture; no exudation.

Henry P—, æt. 32, clerk to a solicitor in the City, was admitted under my care into Guy's Hospital December 23rd, 1857. A tall, well-made, rather pallid, but otherwise healthy-looking man, suffering from entire paraplegia of the lower extremities and sphincters, but without affection of sensation. He stated that he had never previously had any serious illness, but that two years ago he fell whilst attempting to jump over some chairs. After a few days, all apparent effects of this accident passed away, and he considered himself in unimpaired health. In the summer of 1857 he married, and gave himself to excessive indulgence in sexual intercourse; he was otherwise temperate. For two or three months preceding the sudden development of the paraplegia, he experienced at times some difficulty in micturition. The urethra was healthy (1). On the 9th of December there was numbness of the lower extremities, extending as high as the knees (2); but this was so slight as not to attract any attention at the time. On Monday, the 14th, he walked, as usual, from the suburbs to his business in the City. About the middle of the day, as he was crossing his room, his legs suddenly became weak, and he would have fallen had he not been supported. After a short time he recovered sufficiently to walk with some difficulty to the omnibus, and afterwards from the omnibus to his home. In the course of the afternoon he became entirely paraplegic (4, 5), the urine and fæces passing involuntarily from him (7). There was no affection of the upper extremities, except slight and transient formication in the hands (3, 11).

On admission on the 23rd there was only a trace of excito-motor activity in the left leg, and none in the right (6). There was no appreciable diminution of sensation. Movements in the chest normal; pulse 120, feeble; pupils dilated; surface of trunk and upper extremities warm and perspiring; legs cold; a sense of tightness around the chest, about the attachment of the diaphragm (10); bowels inactive; urine drawn off by catheter, acid.

The day following his admission there was noticed to be some œdema of the integuments in the lumbar region, especially on the right side. On the 26th this had almost disappeared. The spine was normal; no change in the paralytic symptoms (14). Occasional slight involuntary twitchings of the legs; electro-contractility of the muscles good; only the slightest trace of excito-motor action, and that limited to the left leg (6). The integuments over the sacrum reddened; pulse 130; skin hot and dry; urine ammoniacal, and containing a large quantity of very offensive mucoid pus; the passage of the catheter was followed by much bleeding. During the night of the 28th nausea and vomiting came on, with great prostration; respiration thoracic. Death from exhaustion on the morning of the 30th, the case having been brought to a rapid termination by the supervention of acute peritonitis upon inflammation of the bladder. The upper extremities were unaffected throughout, with the exception of the slight and transient formication noticed above.

URINARY PARAPLEGIA.

1. Preceded by an affection of the bladder, the kidneys, or the prostate.

2. Usually lower limbs alone paralysed.

3. No gradual extension of the paralysis upwards.

4. Usually paralysis incomplete.

5. Some muscles more paralysed than others.

6. Reflex power neither much increased nor completely lost.

7. Bladder and rectum rarely paralysed, or at least only slightly paralysed.

8. Spasms in paralysed muscles extremely rare.

9. Very rarely pains in the spine, either spontaneously, or caused by pressure, shock, warm water, ice.

10. No feeling of pain or constriction round the abdomen or the chest.

11. No formication, no pricking, no disagreeable sensation of cold or heat.

12. Anæsthesia rare.

13. Usually obstinate gastric derangement.

14. Great changes in the degree of the paralysis, corresponding to changes in the disease of the urinary organs.

15. Cure frequently and rapidly obtained after a notable amelioration of the condition of the urinary organs.

Many theories have been proposed to explain the paraplegia which follows urinary affections. Mr. Stanley, to whom we are indebted for the earliest paper on the subject, supposed it was by the sensitive nerves transmitting a morbid impression to the cord, which was thence reflected outward upon the muscles. Dr. Graves adopted much the same view. He says, "If an irritation of a nerve at the extremities can produce disturbance and exaltation of all the voluntary muscular system, as in tetanus, it is not surprising that such a local cause, tending to depress instead of exalting the motility, should affect not only the nerves and muscles of the part upon which it is developed, but also those of the whole body, or chiefly distant organs."

In 1856 some cases were recorded in the 'Medico-Chirurgical Transactions' which proved that urinary paraplegia was, in some cases at least, due to inflammatory conditions of the cord. The table given above is formed upon the assumption that such cases are exceptional, and that urinary paraplegia is essentially different from that produced by myelitis. The theory of its author is that the paralysis arises from a contraction of the blood-vessels of the cord, nerves, or muscles, set up by a reflex impression starting from the urinary organs; that this reflex contraction of the capillaries, if of any duration, affects the nutrition of the textures. According to this theory, it follows that urinary paraplegia is due to a partial anæmia of the affected parts; whilst if it be due to myelitis or its allied conditions, there is no doubt hyperæmia. The theory is fairly based on the fact, established by recent experiments, that blood-vessels contract with energy, and, to use Dr. Brown-Séquard's own words, are sometimes seized with a prolonged spasm, when the sympathetic which supplies them is directly or indirectly irritated. Thus far, therefore, we have a true foundation. But further than this, in the practical application of the theory, it fails: for it has not been proved to be applicable to the cases before us; and if it had been so proved, the sufficiency of such vascular contraction to produced permanent paralytic effects might be questioned.

That some blood-vessels stand in a nearer relation to nervous influence than others seems to be obvious in the ordinary phenomena of blushing; therefore, until we have more proof than at present, we have good reason for doubting if the blood-vessels of the cord are liable to changes in the same degree that we witness in experiments on the integuments of animals:—whether, in fact, the grey matter of the spinal cord varies in its vascularity in the same degree as the face of a man under emotion, or the ear of a rabbit when the cervical sympathetic is the subject of experiment. Dr. Brown-Séquard would have us answer these doubts in the affirmative. "*I have seen*," says he, "a contraction of blood-vessels in the spinal cord (in the vessels of the pia mater) taking place under my eyes, when a tightened ligature was applied on the hilus of the kidney, irritating the renal nerves, or when a similar operation was performed on the blood-vessels and nerves of the supra-renal capsules." He further adds, "Generally in those cases the contraction is much more evident on the side of the cord corresponding with the side of the irritated nerves: which fact is in harmony with another, and not rare one, observed first by Comhaire (as regards the kidney), and often seen by me after the extirpation of one kidney or one supra-renal capsule; that is, a paralysis of the corresponding lower limb.

The evidence in favour of a reflex paraplegia, thus supposed to be obtained by experiment, vanishes under the criticism of further experiment.

On laying bare the spinal cord and membranes in dogs and rabbits (the animals selected by Dr. Brown-Séquard), no other vessels are visible in the dorsal and lumbar regions than small veins, namely, the dorsal vein and its tributaries. The columns of the cord are seen white and glistening through the membranes. There is not any structure which in itself deserves the name of pia mater, such as exists over the convolutions of the brain. Nor are the columns of the cord much more vascular than tendons. There are no visible vessels on them, nor in the membranes, except those which have been mentioned. It was, therefore, a matter of surprise that these vessels should have been seen to contract; and on placing a ligature on the vessels of the kidney, and irritating the renal nerves, certainly no visible effect was produced—the vessels under observation underwent no change. If our surprise was great at the distinctness with which it had been stated that the vessels of the membranes generally had been seen to contract when the renal nerves were irritated, it was greater still at the further remark, "that the contraction was more evident on the side of the cord corresponding with the side of the irritated nerves;" for such vessels (venules) as are visible in the membranes of the cord are so distributed as to render it apparently impossible that those of one side only should be made to contract. They ramify without any limitation to one side of the cord or the other.

Further, the occurrence of paralysis in the lower limb corresponding to that side on which the kidney was irritated, or the supra-renal capsule removed, appears to have nothing whatever to do with reflex paralysis. Such a result does not follow if care be taken not to injure the lumbar or psoas muscles, in exposing the kidney, and putting a ligature upon it. We found that the animals experimented on used both lower extremities equally well after the extirpation of one kidney or the other. As they walked, we could not in the least conjecture from their gait which kidney had been removed. After this careful inquiry we must say we can discover no evidence, from experiments on animals, for a theory of reflex paraplegia.[1]

Upon the terms of the theory, the animals experimented upon ought to have been paraplegic in one or both extremities, but this was not the case. It may be objected that it would be necessary to repeat such experiments indefinitely, before we could fairly deny that a reflex paralysis occurs. To which it might be justly replied, that if paralysis were only so rarely and exceptionally induced, and the supposed contraction of the blood-vessels, upon which it is said to depend, were only casually to be observed, and not susceptible of demonstration, then we must hesitate before we embrace a theory which rests upon such uncertain and exceptional conditions.

We dismiss therefore as invalid, the proof from experiments on animals that urinary paraplegia arises from a reflex impression on the blood-vessels.

On surveying the clinical history of the disease, it appears to us to accord but little with our knowledge of functional capillary disturbance. In most cases the paralytic affection shows itself only after the disease of the urinary organs has existed months or years; the exceptions to this rule being cases of acute gonorrhœa, which elsewhere have been shown by us to depend upon myelitis. If reflex irritation had been the cause of urinary paraplegia, we should have expected it to occur most frequently at the outset of a case, when the affected mucous surface was most impressionable, rather than after chronic disease had lasted for a long period.

There is one case which is so often quoted in support of a reflex theory, that it deserves a word of mention here. It is recorded by Dr. Graves.

A man, æt. 38, was admitted into the Richmond Hospital in 1835. In the year 1826 he injured his back, and was confined to bed twelve days. In 1830 he was exposed to cold and wet during a long and fatiguing voyage, and began to complain of pain in the lumbar region. At this time he had hæmorrhoids and irritable bladder. In 1834 he suffered much from cold, wet, and fatigue, during a voyage from Cadiz to Dublin, and on his arrival was extremely weak. After resting a fortnight, and living rather freely, he again went to sea; but after nine or ten days his feebleness and the pain in the back increased, so that he was obliged to quit his occupation. When admitted into the hospital his general health was much deranged. He had transient rigors, followed by pyrexia. There were both dysuria and incontinence, and paralysis of the lower extremities. After being treated for ten days by cupping, moxas, demulcents, and opiates, a very narrow stricture was found in the membranous portion of the urethra. A few days after this was dilated, the patient recovered power in his lower extremities in a remarkable manner. It was this sudden recovery which has been regarded as a proof that the paralysis depended upon some reflex impression on the cord from the contracted urethra. How little such an inference can be safely drawn will appear when we consider that the stricture was probably of old duration, and that the paralysis came on only after retention of urine when the body was greatly enfeebled, and that it was relieved so soon as the patient was able freely to empty the bladder. The paralysis appears to have been more connected, in this case, with vesical distension than with the urethral irritation; for certainly this latter existed for a long time before the paralysis. If we admitted the principle sought to be established by those who quote this case, we should certainly have expected to meet with paraplegia from such a cause much more frequently. Urethral stricture is so common, that we ought scarcely to need the evidence of a rare case, occurring once in many years, to elucidate its operation as a cause of reflex paralysis, if it had any true title to be so regarded.

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[1] In these experiments I had the assistance of Dr. Pavy and Mr. Durham, to whom I owe my best thanks for their time and trouble.

The sex of the patients most liable to this disease is not less conclusive against its depending upon mere reflex irritation. Calculus, cystitis, and pyelitis are not unfrequent in women, and yet urinary paraplegia is in them extremely rare. The cause of this immunity is not to be found in their having a less susceptible nervous system, which the reflex theory would seem to require, but in the less complicated anatomical conditions of their urinary organs, and consequently a less need of mechanical interference, and a less liability to the extension of inflammation to the pelvic structures and to the cord.

We have already noticed that children are not commonly the subjects of urinary paraplegia; yet at no age is the nervous system more susceptible.

If we regard the nature of the urinary disease which most commonly leads to paraplegia, we shall find that it is an inflammation, either in the prostate, bladder, or kidneys; and we shall also find, as above stated, that it is only after chronic inflammation has lasted a long time that the paraplegic weakness supervenes. It is in just those cases where there is most irritation, and but little inflammation, that paraplegia does not occur. Uric acid and oxalate of lime calculi may cause hæmaturia and any amount of irritation, but unless *suppurative* inflammation sets in, paraplegia is not produced. A review of all the recorded cases of urinary paraplegia will show that it is the *inflammatory* condition of the urinary organs which leads to paralysis, and not one of irritation.

One conclusion from all these facts is, that urinary paraplegia arises from an altered nutrition of the cord, due to extension of disease through continuous structures to the cord itself. To this it has been objected that we ought in such cases to find traces of myelitis after death; and that if it were a myelitis, we could not expect any improvement in a case of urinary paraplegia—that the disease, having once begun, would more or less rapidly advance.

The statement that the cord has been found healthy in these cases has, in fact, no value. We have long learned how fallacious are the ordinary examinations of the cord. It is a structure which may be extensively disorganised by inflammatory action, and yet have a normal appearance to the naked eye. It has been said that I have myself supplied proof that urinary paraplegia may occur without any change in the cord. The case referred to, however, was not one of urinary paraplegia, as we have seen, but one of asthenia.

How difficult it is to ascertain whether the cord be healthy or not, will be admitted by all who have devoted time to investigating its morbid conditions; and they will be but little disposed to accept any positive statement respecting it, which is not based upon an expert examination by the microscope.

A young married woman was, a few months ago, admitted under my care with entire paralysis of the arms, the legs not being at this time so much affected, though weak. She died from asphyxia, from paralysis of the intercostals. The limitation of the disease to the cervical segments during the early part of the case was most striking. After death the unassisted eye could discover nothing wrong, and, in fact, it was only after a careful preparation of the cord by hardening and thin sections, aided by a gentle daylight, that the degeneration could be made out. I laid the slides on one side at first in despair.

The second objection, that if urinary paraplegia were due to any form of myelitis we could not anticipate improvement, is answered by our daily clinical experience. In the recovery from paralysis caused by antero-posterior curvature of the spine, and after injuries to the back, we have evidence that the cord, like other structures, is capable of repair. This is further confirmed by experiments on animals.

If we inquire into the effects of remedies in urinary paraplegia, it will appear that it exhibits no such sudden and extreme changes as indicate a mere vascular disturbance as its cause. In a large number of cases (excepting such as are not paraplegic, but where there is only debility) the disease slowly progresses. Where improvement shows itself, it is generally the effect of such means as are useful in chronic inflammation of an atonic character elsewhere; namely, careful regimen, tonics, opiates, warm baths, and thermal springs. The subjects of urinary paraplegia are generally enfeebled. It is often the result of sexual intemperance and its concomitants. They who adopt the theory, that the disease is for the most part only a vaso-reflex disturbance, advise on such grounds the frequent use of catheters. This course is not unattended with danger. There is no part of the treatment which calls for more discrimination. The diseased textures and veins about the neck of the bladder are so prone to suppurate, that the catheter is often a fatal weapon. The few scattered instances, such as that recorded by Dr. Graves, where immediate good effects have followed, have had undue influence towards promoting mechanical interference. Carefully considered, they do not warrant the inference drawn from them. If the urinary passages are so contracted that the bladder cannot empty itself, the catheter is obviously required, but it cannot be simply prescribed on other grounds. The rule for its use is the same as in the treatment of the aural passages when the middle ear is diseased. If there be a free exit for the secretions, the less mechanical interference the better. As meddlesome midwifery is bad, so is the meddlesome employment of the catheter in urinary paraplegia. Cases might be quoted where a fatal issue has been induced by this meddlesome interference with a diseased bladder, under the hope of removing some hypothetical cause of reflex irritation.

Since urinary paraplegia has lately been used as synonymous with reflex paralysis from disease of the urinary organs, it is worth inquiry what is meant by "reflex paralysis." The term has been variously used. Dr. Brown-Séquard employs it, as we have done, to express the result of a reflex contraction of the blood-vessels upon the nutrition of the several structures concerned in muscular action. According to others, it signifies a loss of excito-motor activity from an insusceptibility of the incident surface to impressions; as, for example, when dysphagia follows anæsthesia of the fauces. This is, in some sense, a reflex paralysis, or rather a paralysis of the reflex actions. More commonly the term is used to express the progress or transference of a morbid action from the periphery to the nervous centres, and thence on to the motor nerves and muscles by a continuous change in the nerve-trunks or other intervening textures. This is "creeping palsy," or, as it is sometimes called, "peripheral palsy." As illustrations of it, we see injury to a finger involve the whole hand, or even the whole arm in paralysis; or injury to an extremity on one side affect that of the opposite side. The records of medicine contain many such examples. The steps of the process are not always the same. Sometimes the lesion seems to spread along the nerve-trunks by chronic inflammation or atrophy, at other times the blood-vessels (arteries) become slowly obliterated.

A young lady wounded her finger. The arm became generally painful and cold. When brought for examination the whole arm was powerless and wasted. The radial pulse was not to be felt; and on tracing the course of the artery up to the axilla it was found painful and resisting, but without pulsation. By keeping the arm warm, and by time and rest, the power to some extent returned with returning circulation; but, though three years have passed, it has never recovered its full size and strength.

The observations of Waller on the degeneration of nerve-tubules after injury to the nerve-trunks, and those of Turck into the progressive degeneration of the tubules in the nervous centres, when a lesion has begun at any part, promise to throw some light upon what is yet obscure in these creeping palsies. We refer to them only to remark they are not due to reflex action or reflex paralysis; and it is not unnecessary to make this remark, seeing how often the error is committed of assigning every form of palsy which follows a peripheral injury to a reflex effect.

In confirmation of the views here expressed, we quote the opinion of Romberg. He says, "The progress of reflex immobility into paralysis is not as yet proved either by experiment or clinical observation. My former admission of reflex paralysis, against which on many sides doubt and opposition have arisen, has not a secure basis. Since Stanley published the first cases to prove the dependence of paraplegia upon disease of the kidneys, the number of these observations has multiplied, especially by French physicians, but without a corresponding criticism. Not to mention the unsatisfactory description for the most part given of the paralysis itself; the want of evidence afforded by electricity; and the vague indication, noted in many of the cases, of weakness of the extremities; it has been inferred from the apparent integrity of the cord on a post-mortem examination that the paralysis was due to degeneration of the kidneys. It is, however, of little value to assert integrity without microscopic examination (which is not named in any one of the cases), or without having proved that in neither of the columns of the cord nor in any other part was there degeneration."[1]

To this it may be added, in conclusion, that as yet no case of urinary paraplegia has been fully examined, post mortem, without finding such degeneration.

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[1] 'Nerven Krankheiten des Menschen. Dritte veränderte Auflage,' 1857.

LESION OF THE NERVES IN THE NECK

AND OF THE

CERVICAL SEGMENTS OF THE CORD AFTER "FAUCIAL DIPHTHERIA."[1]

During the last two years it has occurred to me to see diphtheria in a severe form in several instances. There have been two varieties of the malady. In one, the disease began in the tonsils or soft palate, and was characterised by an exudation of brownish fibrin upon these parts, often to a very remarkable extent. In some cases, as the disease progressed, the glands and cellular tissue of the neck became swollen and hard, as in phlegmonous erysipelas; the skin over the affected parts became purpurous, and a bloody ichor was discharged from the nostrils. This variety of the disease might be designated, for the sake of distinction, "faucial diphtheria" to distinguish it from the second form, in which the exudation is on the sides or general surface of the tongue, or the mucous membrane of the cheeks and lips, and, in common with these, over the tonsils and soft palate. In this second variety, death when it occurs, arises as far as I have seen, from a cause different from that in the first; namely, by extension of the disease over the respiratory or digestive mucous surfaces, occasioning symptoms of croup and gastro-enteritis: whilst, in the first or faucial variety, death results rather from asthenia, apparently induced by direct injury to the pneumogastrics and sympathetic system of nerves, through inflammation of the areolar tissue in the neck. It is not, however, my object to discuss these points in the history of the disease, but to call attention to the important complication which occurs in the convalescence from the faucial variety. The symptoms appear to arise from a lesion of the trunks of the nerves about the throat, or from a further extension of it to the cervical portion of the cord.

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[1] Reprinted from the 'Lancet,' July 3rd, 1858.

About a fortnight ago I was called to see a boy, of whom I received the following history:—Æt. 11. Had had an affection of the throat, from which he convalesced, and was sent into the country for change of air. About five weeks from the time of his being taken ill it was noticed that he did not carry the head erect—it dropped to one side or the other. There was occasional difficulty in deglutition, loss of voice and attacks of dyspnœa threatening asphyxia. In a day or two from the beginning of these symptoms the breathing became entirely thoracic. The diaphragm was unmoved in inspiration and depressed in expiration, indicating a loss of power in the phrenic nerves. Deglutition was next to impossible. The child could utter no sound. There were fearful attacks of strangulation when the head was moved in particular positions, and even when the breathing was at the best there was blueness of the lips and tracheal râles. The intelligence remained unaffected. The legs could be moved only feebly; the movement of the arms was not impaired; the muscles of the neck were wasted and flaccid; there was no swelling of the fauces; over the transverse processes of the cervical vertebræ, on the right side, there was tenderness, and the adjacent deep-seated absorbent glands were slightly enlarged. No febrile excitement. Pulse feeble, 90. A paroxysm of suffocation suddenly ended the case a few hours after my visit. No post-mortem examination could be obtained.

A few days after I had seen this case a similar one was brought to me by my friend Dr. Kingsford, of Clapton. The previous history was all but identical with that just given, except that the faucial affection had been apparently more severe.

A child, æt. 3 years, had had a severe attack of diphtheria about the fauces, and was recovering when, at the end of a month, it was noticed to carry its head stiffly; the gait was unsteady, the right leg being weaker than the left. Voice husky and indistinct, and deglutition sometimes difficult. The sphincters continued to act well. There were no remains of the disease on the mucous membrane of the fauces. The throat had healed well, but still the deep-seated absorbent glands on the right side of the neck could be felt enlarged. The movements of the chest and upper extremities were normal.

The extension of inflammation from the contents of the pelvis to the membranes and lumbar segments of the cord has been elucidated by some cases of paraplegia recorded in the 'Medico-Chirurgical Transactions' for 1856. These cases following diphtheria appear to belong to the same pathological series, the cervical portion of the cord being affected from disease in the fauces in a similar way to the lumbar segments in vesical or other pelvic affections. They are so far imperfect that the first was not examined post mortem, and the second is still under treatment, but they appear to be sufficiently distinct as illustrations. I should not have ventured to record them, however, without further confirmation, but that it seemed desirable not to delay an inquiry where, it is to be hoped, the opportunity for further observation will soon have passed away with this new or newly revived pestilence.

CASE

OF

PROGRESSIVE ATROPHY OF THE MUSCLES OF THE HANDS:

ENLARGEMENT OF THE VENTRICLE OF THE CORD IN THE CERVICAL REGION, WITH ATROPHY OF THE GREY MATTER (HYDROMYELUS).[1]

THE following case is given as a contribution to the pathology of "progressive muscular atrophy."

Three classes of cases are as yet confused together under this term and its synonym, "wasting palsy."

Progressive, or perhaps better, "excessive" muscular atrophy, may arise from primary lesion of the muscular elements—or from lesion of the trunks or branches of the nerves—or from morbid changes in the grey matter of the cord. It is the difficulty of distinguishing the primary seat of disease in each of these classes of cases, which has led to exclusive, and therefore erroneous views of their pathology, one observer maintaining that progressive muscular atrophy is always a peripheral affection, whilst another asserts that it has constantly a centric or spinal origin.

Besides the forms here enumerated, there is a fourth class, in which muscular nutrition fails from a morbid diathesis allied to rickets. It has nothing in common with the pathology of the former cases but the want of muscular power. Dr. Meryon's[2] are good examples. I have seen the disease in two girls of one family. It is a malady beginning in, and limited to childhood, and peculiar to it. The muscles are not diminished in bulk at the commencement of the disease, nor is there necessarily any paralysis during the whole course of the case, but only a lentor and feebleness of movement. This cursory mention of such cases is sufficient to show with what little practical value they can be included in the first enumeration. No doubt much of the obscurity which at present besets this subject is favoured by the assumption, that where disease was not discovered in the cord, it did not exist; happily, however, positive assertions from negative evidence are at this day considered of less weight. We cannot peruse recorded cases of progressive muscular atrophy, without feeling how unsatisfactory, in most of them, is the post-mortem examination of the cord, and must hesitate to accept the conclusion that it was free from lesion, because none was discovered. Dr. Beale's 'Archives' (No. 9) contain an almost critical case in illustration of these remarks, and certainly, but for the rare skill of Mr. Lockhart Clarke, it might have been recorded as one of muscular atrophy, the cord being healthy. Thanks, however, to the means we possess of investigating ultimate structure, Mr. Clarke was able to show that there were certain areas of the grey matter which had undergone marked change of a morbid character, although the cord had an entirely healthy appearance. During the life of the patient referred to, there was a difference of opinion as to the primary seat of the malady, and but for such an exhaustive examination as it received after death, instead of its being a contribution to our knowledge, the record of the case would have served only to give strength to false assumptions, and fortify us in the error of assuming that all is sound where imperfect examination detects no weakness.

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[1] Reprinted from the 'Guy's Hospital Reports,' 1862, p. 244.

[2] 'Medico-Chirurgical Transactions,' vol. xxxv, p. 73.

The following case, on its entrance into the hospital, gave rise to the same question—whether the muscular atrophy had a peripheral or central cause. The patient was a journeyman tailor, working hard at his business in London, and, therefore, of necessity using the muscles of his hands in an extreme degree. Here, therefore, was a possible, and not an improbable cause of a primary affection of the muscles themselves, but, as was remarked in a clinical lecture given on the case, it was to be remembered that the will does not directly act upon the muscles in voluntary movement, but upon the grey matter of the cord to which the nervous filaments are connected, and therefore, that it is as reasonable to infer a lesion of the grey matter from overwork, as of the muscles.

The first symptom, in this case, was inability to extend the little and ring fingers of the right hand, with a sense of coldness and numbness in the part. This was the only complaint for eleven months, and no doubt, if the patient had been seen during that time, it might have been thought more probable by some that the disease lay in the muscular tissue than in the centre of the cord. It was not until after eleven months that the adjoining middle finger began to fail in a similar way. A perusal of the case will show, that the centre of the cord had by this time undergone extensive changes, yet it was not until the left hand became affected, that the central character of the lesion began to appear, and even then it might have been contended, that the symmetry of the muscular affection was owing to the same conditions of over-wear in both hands. Had it not been for typhus fever, which then prevailed with unusual virulence in the hospital, we should not have known how great morbid changes the central grey matter of the cord may undergo, with but slight and limited and only slowly progressive peripheral effects. The lesion discovered after death was evidently in no way connected with the attack of fever, nor in any way affected by it. The tissues at fault showed no traces of recent activity about them. This case is therefore another instance of atrophy progressing from muscle to muscle in the slowest way, and unattended by any of the common proofs of central disease, though depending upon it.

It also suggests some doubt respecting the validity of the present theories of the function of the grey matter of the cord. A glance at Plate IV (p. 282) of a transverse section of the cord in this case, will show how large a part of the grey matter may be slowly removed, without affecting sensation to any corresponding extent, and without disturbing the general functions of the cord, or the influence of the brain upon the parts below.

Although there were no other remains of the grey matter in certain parts of the cervical region except the anterior cornua, the patient was still able to walk perfectly well, and to move the arms freely in all directions, and the sphincters were good, nor was there any affection of sensation in any part, except a feeling of numbness in the right hand.

What the nature of the change in the cord was, may be a matter for speculation. So far as it affected the grey matter, it seemed to be no more than atrophy from distension of the ventricle of the cord, by an accumulation of fluid in it—a chronic cervical *hydromyelus*, comparable to a chronic *hydrocephalus*.

It is noticeable that normal epithelium still lined this extremely dilated ventricle.

The appearance of a distinct membrane to the cavity was produced by condensation of the normal textures pressed outwards, and not by any new plasma.

The extent to which the disease reached in a longitudinal direction, is shown in the woodcut, the dotted outline in the middle of the cord indicating the extent and form of the cavity in the interior.

The greatest diameter of the cavity was opposite the origin of the seventh or last cervical nerve, and hence the peripheral effects were chiefly marked in the branches of the ulnar nerve, which here has its principal origin.

The form of the cavity, on a transverse section, is shown in the plate. It will be observed that it was not a simple circular dilatation, but corresponded to the general disposition of the grey matter and its cornua.

G. B—, æt. 44, a journeyman tailor, of sober habits, was admitted into the clinical ward, February 5th, 1862. States that he has always been healthy and strong. Never had any injury to his back. Thirteen months ago, when working in London, the fourth and little finger of the right hand became weak and flexed, without any assignable cause. The hand was cold, and there was a feeling of numbness in the fingers, but no pain. This gave rise to a good deal of inconvenience, but he was able to continue working at his trade. Two months ago the middle finger of the same hand became suddenly affected, and three weeks ago the three inner fingers of the left hand became weak and flexed in the same way, but without any numbness. The hands gradually wasted. The arms are not affected. Seven weeks ago he had pains through his chest, and a feeling of tightness across the upper part. He is pale, complains only of wasting and weakness of the hands; has no pain in them, but the right is cold, with a feeling of numbness. The left hand is not so cold, and the sensation in it is perfect. He can move both thumbs and index fingers freely; he can also extend the first phalanges of the other fingers of both hands, but not in the least degree the second and third phalanges, which are gently flexed towards the palm. The interosseous spaces on the backs of the hands are sunken from the wasting of the muscles. The palms of the hands are hollow, and the flexor tendons very prominent. The thenar eminences are wasted, and the hypothenar almost entirely gone, particularly on the right side. The motion of the wrist-joints is unaffected. He can move the arms freely in all directions. Can walk perfectly well.

At the upper part of the dorsal region there is slight flattening of the natural curve of the spine, from the long muscles of the back being at this part wasted. Pressure on the fourth dorsal spinous process causes a sharp pricking pain, as of a knife running into the part, but when the part is not touched he has no pain. No pain on pressing the other spinous processes; no affection of sensation in any part except the feeling of numbness in the right hand; sphincters good; urine normal; appetite and digestion good. He was put upon a full diet, and the wasted muscles were daily galvanised by an intermittent current. A fortnight after admission he had gained power in the hands. He said he felt them stronger and more pliable after each application of the galvanism. It was noted that, with a moderate current, the contractility of the muscles of both hands was good, but more particularly in the short muscles of the thumb, which were least wasted. Sensibility not so acute in the right hand as in the left, but no marked anæsthesia of either. Both hands were rather cold. A few days after this report the patient sickened with typhus fever, and died on the 8th of March

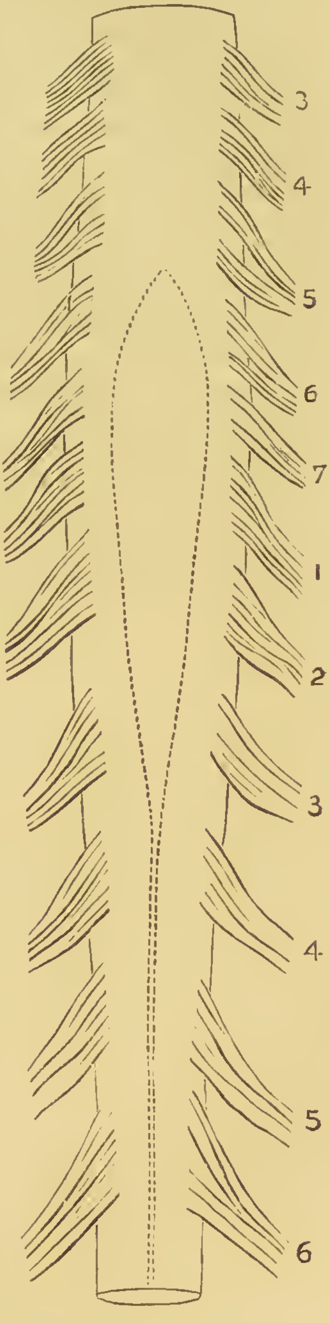


Diagram of spinal cord showing enlargement of ventricle.

A post-mortem examination was not permitted at the hospital, and it was only after much difficulty that the cord could be obtained. The bones and ligaments of the spine were healthy; the membranes of the cord healthy; the exterior of the cord presented nothing abnormal, except that the cervical enlargement appeared broader and somewhat flattened. On making transverse sections the white columns had their normal consistence and texture, but the centre of the cord had a large cavity, beginning at the fifth cervical, enlarging downwards to the seventh, and from thence tapering as in the accompanying woodcut. The appearance of the cord on a transverse section at the origin of the seventh cervical nerve is shown in the annexed plate. It will be seen that the only remains of the grey matter are at the anterior part of the cavity behind the anterior columns. Here the caudate vesicles had their normal size and structure; the pigment, nucleus, and nucleolus being well marked, and the tubular structure unaltered. The cavity in the cord was bounded by a layer of condensed grey substance, which could be separated as a distinct membrane. On its interior surface, forming the lining of the cavity, were a number of delicate, elongated, nuclear bodies, apparently epithelium. One or two granule-cells were found scattered amongst the white columns, but no further traces of any active tissue change. The roots of the nerves appeared normal, and contained healthy tubules. The character of the fluid filling the cavity could not be ascertained, as it escaped in the removal of the cord from the spinal canal.

PLATE IV.

Transverse section of the spinal cord, at the origin of the seventh cervical nerve (Case of G. B—, p. 280), showing enlargement of ventricle.

The centre of the cord was hollowed out into an irregular square cavity or ventricle, with cornua corresponding to the general disposition of the grey matter.

This cavity was limited by condensed grey matter, and lined by an imperfect epithelium.

Towards the posterior columns, and at the origin of the posterior roots, it will be seen that little, if any, grey matter was left.

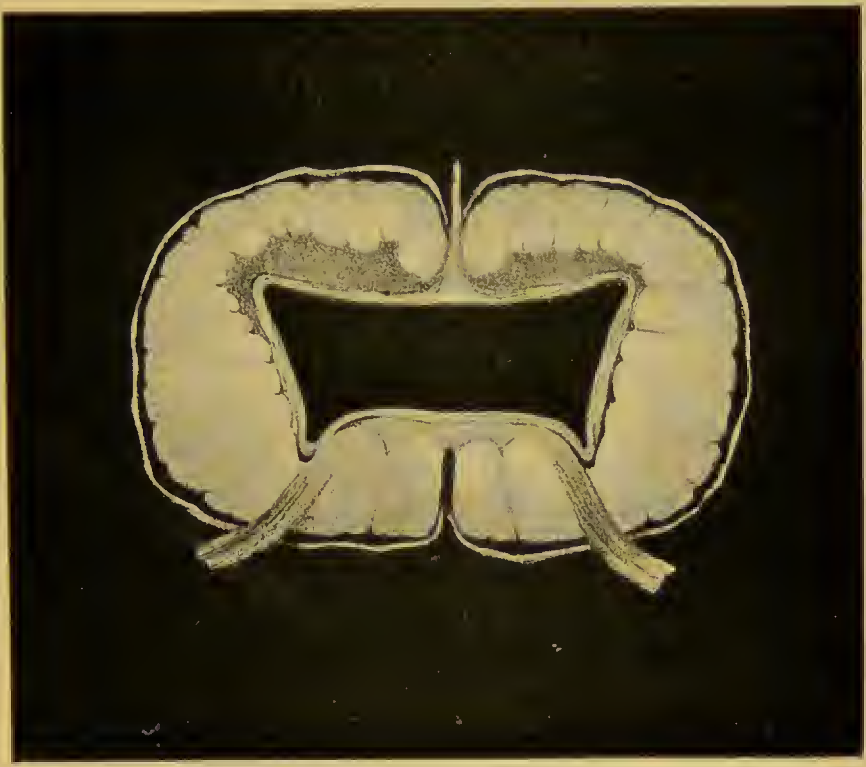
Anteriorly the grey matter is spread out, and in it, by the microscope, the caudate vesicles could be seen, having their normal structure.

The white columns are shown in the drawing a little disintegrated, and irregularly spread out, owing to the thinness of the section.

They had quite a normal structure, except that after repeated examination, one or two granule-cells were detected in them.

The membranes surrounding the cord, shown in outline in the plate, were healthy.

PLATE IV.



Case G. B—, p. 280.

Adlard and Son, Imp.

(*c*) DISEASES IN WHICH SYMPTOMS REFERABLE TO THE NERVOUS SYSTEM ARE PREDOMINANT.

HYPOCHONDRIASIS.[1]

Definition.—A disease of the nervous system, of unknown and possibly varying seat. It is markedly hereditary, being one of the transformed neuroses which descend from a parent stock strongly tainted with insanity. Its principal feature is mental depression, occurring without apparently adequate cause; and taking the shape, either from the first, or very soon, of a conviction in the patient's mind that he is the victim of serious bodily disease. The sufferer's belief in this disease is so firm, that he describes minutely the symptoms, which as he fancies, indicate its existence. But he may place the imaginary malady in almost any organ of the body, and he usually describes some symptoms which are anomalous, or even incredible. Finally, hypochondriasis may be evoked by a real organic disease acting as an irritant to an hereditarily predisposed nervous system; in this case, the anomalous nervous symptoms may mask, and even conceal, the occurrence of serious changes in some viscus.

Nomenclature.—The same name has been applied to the disease since the days of Hippocrates: it has always been known as "Hypochondriasis," or the "hypochondriac disorder," and sometimes as the "vapours," or the "spleen;" but these two last synonyms are, in fact, mere explanations of the ancient hypothesis which was expressed in the word Hypochondriasis. This hypothesis it is really important to say a few words about, since the vulgar conception of the disease still, though unconsciously, cherishes it; although our improved knowledge of the relation of the nervous system to the organism generally has now made it an anachronism.

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[1] By William Withey Gull, M.D., and Edmund Anstey, M.D. Reprinted by permission of Messrs. Macmillan and Co. from Reynolds' 'System of Medicine,' vol. ii, 1868, p. 293.

The words ὑποχονδριακὸν πάθος, applied by Hippocrates and Galen to the disease, imply a belief that the viscera behind the xiphoid cartilage, and below the diaphragm,[1] were its seats; and Galen very distinctly says that *black bile* is its cause. It is worth while to recall for a moment the physiological ideas which Galen, with great ingenuity, had compounded from the speculations of Plato on the one hand, and Hippocrates on the other. According to Galen, the functions of the human body were maintained by three πνεῦματα (whence, remotely, our "vapours"). The lowest of these was the πνεῦμα φυσικὸν, and developed the *natural force* in the liver; the second was the πνεῦμα ζωτικὸν, which elaborated the *vital force* in the heart; and the third and highest was the πνεῦμα ψυχικὸν, which developed *animal* or *soul force* in the brain.[2] Anyone who has been curious enough to investigate these questions will see at once that the lowest or "natural" force of Galen is the counterpart of that lowest kind of *mortal soul* which Plato[3] represented as residing in the abdominal organs, and chiefly in the liver, and as having to do with the baser animal passions and the supply of the needs of vegetative life. The ancient tendency to view every source of functional activity as an entity—almost a personality—made it quite consistent for the long succession of Galenist physicians to endow the liver-force with a quasi-consciousness and perception, and even with voluntary activity, though of a low kind; and, on the other hand, the Paracelsian and Helmontian doctrine of the *Archæus* rather added to than changed anything in the extraordinary power over the general organism which was attributed to the abdominal organs. Then the absence of any accurate knowledge of the functions of a central nervous system, the recipient of sensory impressions, and the originator of motor acts, induced men to localise in the various organs the source of the functional disturbances which appeared to be manifested therein. The vagaries of hypochondriacal sensation were therefore, in the ancient view, the perturbations of the natural force generated in the liver, spleen, and pyloric part of the stomach. It is to be remarked, moreover, that hypochondriasis was very generally confounded with hysteria (to which it doubtless has a relation) quite down to the present century. There is nothing surprising in this. The flatulence which is frequently a striking symptom both in hysteria and hypochondriasis was, for the ancients, a commotion of the natural spirits which resided in the abdomen.

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[1] The viscera of the hypochondria, to which the ancients attached such importance, seem to have been especially the liver, the *pyloric part* of the stomach, the omentum, the mesentery, and the spleen. The stomach considered as a whole they regarded rather lightly.

[2] 'De Loc. Affect.,' v. 'De Usu Part.,' v. 'De Usu Resp.,' 163, 164.

[3] 'Timæus,' ed. Stallbaum, §§ 69, 70.

Of the long list of authors who have treated of hypochondriasis, since the days of Galen, there is scarcely one who viewed the disease in any essentially different light from that in which Galen regarded it, until we come to Thomas Willis, the great investigator of the nervous system. It is easy to see what were the common ideas on the subject at the time by consulting that curious book, Burton's 'Anatomy of Melancholy,' which was published a few years after Willis's death, and which represented the knowledge which a learned and clever layman might pick up without knowing, or without demeaning himself to notice, the writings of a contemporary countryman. Burton says that the general view of authors represents the hypochondriac or windy melancholy as arising "from the bowels, liver, spleen, or membrane called *mesenterium*," and quotes Laurentius as dividing it into three kinds, the *hepatic*, the *splenetic*, and the *mesaraick*.

Willis,[1] on the other hand, placed hypochondriasis amongst the diseases proper to the *diathesis spasmodica ;* he made it an affection of the nervous system, but so far conformed to the old ideas as to attribute its ultimate causation to impurity of the splenic blood. In the next century, Flemying[1] ventured a more distinct opinion that the brain was the part primarily affected, and Cullen[2] and Robert Whytt[3] (especially the former) placed great stress on the share which the nervous system has in the production of the disease. The next prominent step was taken by Georget[4] (1819), who protested against the view (at that time still commonly prevailing) of the abdominal origin of hypochondriasis, and the practice of powerful purgation, &c., which was based upon it. But the most complete and effective attack on the old view was made by J. Falret,[5] in 1822. This author dwelt strongly on the hereditary character of the disease, and the great frequency with which it is immediately excited by stress of intellectual labour, or by moral and emotional causes. The view of Falret was perhaps carried to excess in limiting the primary seat of the disease so strictly to the brain; but it has prevailed, and hypochondriasis is now commonly placed among the varieties of insanity. Griesinger, for instance, in his admirable treatise on mental diseases,[6] makes hypochondriasis a variety of melancholia, which is his first class of "mental diseases characterised by depression;" and Leidesdorf[1] adopts the same view. It will be seen that the view which we hold differs in some degree from this; but there can no longer be any doubt that the true seat of the disease is in the nervous centres.

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[1] T. Willis, 'Opera Omnia,' 4to, Genevæ, 1676. The whole treatise, 'De Morbis Convulsivis,' and that on 'Hysteria and Hypochondriasis' (in reply to the strictures of Nat. Highmore), are astonishing efforts of genius, and will well repay perusal in the present day.

History.—The history of a hypochondriac patient is that of his nervous system under the two aspects of its congenital form and the influences—of nutrition, education, and emotion—to which it has been subjected. So far it is not different from the story of the sufferer from any other form of nervous disease. But hypochondriasis is distinguished by this,—that a more important part is played by the congenital disposition of the nervous system, and a less important part by the physiological and spiritual influences which have been brought to bear on it, than is the case with the majority of nervous diseases. It comes nearest, in this respect, to insanity on the one hand, and to epilepsy and neuralgia on the other. It is the almost *inevitable* inheritance of a certain percentage of the descendants of any individual who may be very strongly tainted with insanity. On this subject we shall say no more till we can discuss more fully the etiology of this singular disease.

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[1] 'Neuropathia, sive de Morb. Hyperchond. et Hyster,' Ebor., 1744.

[2] 'Clinical Lectures,' London, 1777, pp. 39—57.

[3] 'Observations on the Causes, Nature, and Cure of the Disorders called Nervous, Hypochondriacal, &c.,' London, 1777.

[4] 'De la Phys. du Syst. Nerv.,' Paris, 1819.

[5] 'De l'Hypochondrie et du Suicide,' Paris, 1822.

[6] 'Die Path. und Therap. der Psych. Krankheiten,' 2nd edit., Stuttgart, 1861.

[7] 'Path und Therap. der Psych. Krankheiten,' Erlangen, 1860.

Symptoms.—This phrase is pre-eminently appropriate to the phenomena of hypochondriasis. Of physical signs we have almost none to guide us; and this is in perfect agreement with the position which this disorder holds in the category of diseases generally. All is in the region of symptoms. For the most part, too, the symptoms are subjective only; still there are features which the experienced physician can detect, and which can hardly be simulated by a malingerer.

The most important external feature of hypochondriasis is this,—that without any sufficient reason for such conduct, and without any signs of intellectual insanity, the patient is observed to concentrate his attention on some particular organ of his body, and to fancy that it is seriously diseased. This concentration of attention is often preceded and accompanied by notable depression or variability of his spirits, with a tendency, on the whole, to depression; this is not always the case, however, for there is sometimes no antecedent symptom connected with the general mental state. In many instances the patient's first sufferings take the form of what he himself considers dyspepsia, but which is in fact little more than flatulence, from the formation of large collections of gas in the stomach and bowels. Along with this flatulence there are sometimes appearances which give a superficial colour to the idea of a primary stomach derangement; the tongue, for instance, is often pasty and coated, and there may be foul breath; the appetite is not unfrequently ravenous, capricious, or well-nigh lost; there is generally obstinate constipation; in rare cases there are even attacks of vomiting.

More commonly there is an antecedent mental change, the character of which it is at first difficult to seize, and which forms one of the grounds for the modern practice of including hypochondriasis in the varieties of actual insanity. Before any local symptoms have declared themselves, the patient has already become changed in his disposition; in most cases, perhaps, the change is simply in the direction of despondency or vague alarm, for which he can give no reason. It is remarked by alienists that the mental condition, even thus early, is characterised above all things by an expansion of the *self-feeling*, a pre-occupation of the patient with his own condition, to the exclusion of other interests and affections. This is true, but it appears to us that the self-concentration is more like that of a person in the preliminary stage of an acute inflammation or fever, the nature of which is not yet declared, than the egotistic alteration of character which seems to lie at the basis of insanity, and which probably depends directly upon minute changes taking place in the cortical substance of the brain. It is a real bodily sensation (though at first indescribable), which enchains the sufferer's attention; and before long this vague feeling is exchanged for a positive localised sense of uneasiness or actual pain.

Sometimes the early mental state is one not merely or chiefly of despondency, but characterised by suspiciousness and irritability of temper, with quick changes from high spirits and loquacity to moody silence. In any case, after a time, the patient not only exhibits in his aspect and conduct the general uneasiness from which he suffers, but begins to complain of definite subjective symptoms. Probably the most common of these is pain of a gnawing or burning character, or else a sense of great though vague uneasiness at the pit of the stomach. But in fact any part of the peripheral distribution of the sensory nerves may be the apparent seat of painful sensation, and besides this there is very often a general heightened sensibility of the skin. Both the active pain and the heightened sensibility of (uncomplicated) hypochondriasis are subjective, and resemble the similar phenomena which are so common in hysterical women, in vanishing when the patient's attention is power-fully diverted from them. The painful sensations of which hypochondriacs complain are very acute; and their severity concentrates the attention of the sufferer exclusively upon them, increasing the apparent egotism of his disposition. But it may here be remarked that the heightened self-feeling of hypochondriasis does not partake of the despondency of true melancholia, still less of the character of other forms of insane egotism. The patient (as observed by Leidesdorf), though depressed in mind, not only wishes to get rid of his malady, but has great faith that he shall do so; a faith which suffers repeated shocks, indeed, from the non-success of particular remedies, but quickly revives in favour of some new mode of treatment. The eagerness with which he pursues the means of cure is the true cause of the limitation of his thoughts.

Next to pseudo-dyspeptic symptoms, and the occurrence of pains or anomalous feelings at the pit of the stomach, the most common morbid sensations in hypochondriasis are probably formication of the skin, and *burning pains* in the course of particular superficial nerves. It is noteworthy that, so far as we are aware, the nerve-pains most frequently assume the burning type, rather than the lancinating, throbbing, or aching forms which neuralgia more commonly takes. A common occurrence is the sudden shifting of the pains or the sense of formication from one part of the body to another, or their sudden extension from a small area which they first affected over almost the whole surface of the body. Another very frequent subjective symptom is the feeling of pain, or great but indescribable uneasiness, deep in the heart, or the lungs, or the liver, the bladder, or the rectum. The development of the subjective symptoms is very often seriously influenced by the fact that the patient is driven by his misery to consult medical books, or to pester his medical friends with questions bearing on his sufferings; his defective knowledge and distorted fancy lead him to apply, *à tort et à travers*, the scraps of information which he picks up, and to imagine, successively, that he has discovered in himself the signs of one, two, or half a dozen serious organic diseases. Attention being thus directed to particular organs, the subjective symptoms naturally increase and multiply, and the emotional excitement produced also frequently sets up severe functional disturbance, such as flushings of the face, abdominal pulsation, palpitation of the heart, partial suppression of bile and jaundice, or bilious diarrhœa; symptoms which still further confirm the sufferer in the belief that he is labouring under serious organic disease. A very common delusion is the belief that there is fatal heart disease; and a scarcely less frequent one is the persuasion of the patient that he is impotent from spermatorrhœa; this last is of course greatly fostered by reading pseudo-medical treatises. In the case of patients whose family is strongly tainted with insanity, the anomalous sensations often assume a type which approaches to hallucination or illusion (as where there is the belief that a serpent is writhing about in the entrails, &c.), or the judgment becomes affected to such a degree that the patient entertains preposterous delusions (as that he is made of glass and in danger every moment of being broken, that he is being magnetised, that people are conspiring to poison him, &c.). The delusions sometimes are confined, at any rate for a time, to one or two organs, but are nevertheless so extravagant that it would really seem no paradox to say that the patient has a mad stomach, a mad liver, or a mad bladder; while on all other matters his intellect remains healthy, and often unusually acute. But on the subject of the protean symptoms of hypochondriasis it is really unnecessary to enlarge further, and we may refer those who desire to read a truthful and highly picturesque description of them to the pages of Burton,[1] to say nothing of more modern writers.

Diagnosis.—The diagnosis of hypochondriasis, from maladies superficially resembling it, is proverbially beset with difficulties, and the practitioner can only gain confidence in his decision on the more doubtful cases by means of long experience. Nevertheless, the general principles on which his judgment must be formed are not very difficult to state.

If the anomalous character of a patient's subjective symptoms points in the direction of hypochondriasis, the very first subject of inquiry should be the family history. A well-defined history of insanity in the race would at once indicate the probability that the patient's sensations were partly illusory, and not referable to their apparent site. On the other hand, a decided history of the absence of insanity and of the other severe neuroses from the family during two or three previous generations, would still more strongly suggest that the case was *not* one of hypochondriasis. The next point for investigation would be the mode of commencement of the illness. A history of the primary occurrence of severe bodily symptoms, whether in the shape of pyrexia, of disturbance of hepatic or gastric functions, or of pain in the course of nerves, is unfavorable to the diagnosis of hypochondriasis, unless these phenomena were preceded or accompanied by psychical changes such as have been already described. Even a more chronic development of capricious pains, of formication of the skin, of flatulence, palpitations, and the like, is not specially indicative of hypochondriasis, unless there is unusual anxiety on the patient's part, and an egotistic tendency to dwell on his sufferings. A great deal may be gathered from considerations of age and sex. Hypochondriasis is pre-eminently a disease of adult and middle life; it is hardly ever seen before puberty, and it very rarely makes its first appearance after the age of fifty. It is greatly more common among men than among women; in the latter sex it appears to be replaced, for the most part, by hysteria. Still hypochondriasis may occur in women, and the question of diagnosis from hysteria, in such a case, becomes important, and may be very difficult. Beyond all other circumstances which favour the probability of hypochondriasis is the fact of a strong hereditary taint of insanity. The age at which the symptoms commence is important; thus the first appearance of hysterical phenomena nearly always takes place between the ages of fourteen and thirty, or else at the grand climacteric; and has very commonly a marked relation to those changes in the nervous system which correspond with the changes of the sexual apparatus; whereas the development of hypochondriasis is especially associated with the circumstances of middle life—in the rich and idle with the *tedium vitæ* of a purposeless existence; in the poor and anxious with the cares of a family, and perhaps with the added misery of a conscious failure in efforts to support relations and dependants. Severe moral and emotional shocks may be followed either by hypochondriacal or hysterical disorder; but the latter is the infinitely more probable result in women who are not descended of markedly insane families, and especially in women who lead busy lives.

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[1] 'Anatomy of Melancholy,' pp. 270—274.

One of the most important questions in diagnosis is the decision whether, if hypochondriasis be present, there is not at the same time some organic visceral disease; for it sometimes happens that the first sign of the occurrence of such disease is an outbreak of hypochondriacal symptoms, the patient being hereditarily predisposed to the latter disorder, and his nervous system excited to morbid action by the irritation of the new organic processes which are going on. Of the diseases which have been known to produce such an effect, structural changes of the liver, and next to them structural changes of the stomach, are probably the most frequent examples; and, after these, aneurisms of the great vessels, valvular diseases of the heart, angina pectoris. It is unnecessary here, even if there were space, for us to go into the characteristic symptoms of these organic diseases. The first feature which may lead the physician to suspect the existence of organic visceral disease in the midst of symptoms which he feels sure are hypochondriacal, is the persistence of some one complaint by the patient—*e. g.* of pain in a particular locality; especially if this be combined, always in the same order, with other symptoms that belong to the suspected organic disorder, and with which the patient is not likely to be acquainted so as to be mentally influenced to reproduce them. Thus if, along with a fixed complaint of pain in the præcordia increased by swallowing, there is the regular occurrence of regurgitation of some of the food very shortly after deglutition, it is a case for inquiry as to the possible existence of cancerous or other stricture of the cardiac end of the stomach, &c. It is needless to say that physical signs, when they are present, are the most valuable helps in discerning organic disease which is masked by hypochondriasis; yet even here there is need for caution. For instance, the occurrence of hardness and tumidity in the epigastrium or the hypochondrium, in such a form as closely to imitate a scirrhous tumour (even on repeated examinations), may be produced by spasmodic contraction of one or both recti: in such a case the administration of chloroform would at once dismiss the suspicion by dissolving the "phantom" tumour. A circumstance which is always of doubtful interpretation is the occurrence of wasting, especially if combined with jaundice. If this takes place with rapidity, it can hardly be owing to hypochondriacal disturbance of digestion and assimilation, but is probably due either to the generally depraving effect of cancerous or tubercular taint, or to direct interference with nutrition from the mechanical effects of ulcer, stricture, or tumour, upon some of the chylopoietic viscera. The combination of insidious and unsuspected malarial poisoning with hypochondriacal tendencies may produce formidable difficulties in diagnosis, which can only be overcome by careful study of the patient's past history, sometimes by the discovery of enlarged spleen, and above all by the effects of antiperiodic medicines. Another variety of blood-poisoning, which in hypochondriacal patients may be somewhat masked, is chronic alcoholism; but the symptoms of the latter complaint are, after all, tolerably distinct and recognisable from their peculiar grouping, and even in a hypochondriac they may generally be identified.

A more serious difficulty in diagnosis than any which has yet been mentioned is the distinction between certain forms of hypochondriasis and true melancholia. Given a patient with a decided family history of insanity, with a mental condition marked by a strong tendency to dwell on complaints of bodily misery, and with dyspepsia and flatulence, it may be very difficult to say whether or not the case will pass into true melancholic insanity. The following case will give a good idea of the occasional uncertainty. A postman aged forty-three, a widower, was much overworked, and especially harassed by having to sort the letters in the morning before he started on his beat—a task which had to be hurriedly done, and hence gave him much anxiety lest he should make mistakes. He applied for relief from dyspepsia and flatulence and bilious diarrhœa, but at the same time complained that his spirits were dreadfully low, that he had thoughts of suicide, and that he believed he had "something alive in his inside." A simple tonic mixture of mineral acid and bark, combined with the moral influence of encouraging assurances from the physician, did this patient so much good that he soon seemed perfectly well, and remained so for some months. He then got married again, and his marriage apparently embarrassed his means, though not seriously; but his despondency now returned in the form of a belief that he and his family would have to go to the workhouse (of which there was really not the least danger), and the impulse to suicide again became very urgent. At the same time his dyspepsia and bilious diarrhœa returned. He applied for medical relief, was ordered the same treatment as before, and was encouraged to hope for a speedy cure; but the very next day he attempted suicide by completely severing the whole of his genitals from his body with a razor. He was taken to St. George's Hospital, and with great difficulty kept alive while the wound healed. Six weeks after his discharge from the hospital he appeared before his former attendant, looking fat and fresh-coloured, but more despondent than ever—indeed, plainly insane. He was then lost sight of, but there could be little doubt that he would get worse, and, if not carefully watched, would commit suicide.

Such a case as the above has little to separate it from hypochondriasis except the one important feature of the early occurrence of *suicidal despondency*; but this feature would probably be sufficient justification for a decided diagnosis. It is only where the hypochondriac patient has been exhausted by a long continuance of his sufferings and rendered hopeless by the failure of a thousand attempts at cure, that he turns his thoughts to self-destruction, and by that time he may be considered really insane. Indeed, the hypochondriac proper regards the idea of suicide with the utmost repulsion and disgust.

There is no serious difficulty in distinguishing simple hypochondriasis from the other forms of insanity.

Prognosis.—The prognosis of hypochondriasis varies extremely, not so much with the apparent severity of the symptoms, as with the circumstances under which they arose, the length of time during which they have already persisted, and above all the degree to which the patient's family has been tainted with insanity. But in general it may be said that the younger the patient, the shorter the time during which he has suffered, the less that the nutrition of the body has deteriorated, and, above all, the less of decided family taint of insanity that can be traced, the more hopeful is the aspect of the case, and *vice versâ.*

Etiology.—The "causes of hypochondriasis" is an expression even more singularly unhappy than the average instances of a phraseology of causation applied to those circumstances which precede the outward and visible development of functional disorders. To commence with those influences which have a conventional title to be called "exciting" causes, it is undoubtedly true that in a considerable number of cases the train of disastrous events has seemed to be fired by the moral collapse consequent on over-exhausting labour, or bitter disappointment of cherished hopes, or on the sudden revelation to the mind of an idle man that he is a mere burden on the face of the earth. Again, it is commonly said that reading or conversation on medical topics often frightens laymen, and, more rarely, even doctors, into a nervous and hypochondriacal frame of mind. There is doubtless something to be attributed to such influences, but the most thoughtless person could hardly fail to be struck, on reflection, with the surprising infrequency of hypochondriasis in comparison with the ubiquitous operation of such influences as grief, fatigue, the sense of shameful failure, the habit of miserable and heart-wearying idleness, and the practice by the laity of reading medical treatises. If we turn to the events which would be conventionally spoken of as "predisposing" causes, we are scarcely likely to be more satisfied with the appropriateness of the term "cause;" though we come upon facts of far greater practical value than those which have just been mentioned. The fact of hereditary insane taint, for instance, is an antecedent which is observed in an immense number of cases, if not in all. The preponderance of males among hypochondriacs is equally unmistakable, and so is the fact that the bulk of cases occur in persons in the prime and vigour of life. It is also an undoubted fact that the average level of intellect in hypochondriacs is not below, but rather above, the general standard; and that their bodily health has often been excellent up to the moment when the nervous symptoms made their appearance. But instead of saying that these circumstances are "causes" of the disease, it will be convenient to say that they are *conditions* of its occurrence in the following degree and manner. A certain number of the descendants of a family strongly tainted with insanity will invariably be born with peculiarities of the nervous system; these peculiarities do not, probably, consist of recognisable structural faults, but rather of tendencies of one or more (perhaps scattered) portions of the central system, to change interstitially in a certain morbid direction, at particular crises of life, through which healthy organisms pass unharmed. Arrived at such crises the nervous system will surmount them, or will succumb, according to the absence or the presence of certain external disadvantages. If the morbid change occurs, it will not affect the machinery of the intellectual and reflective faculties chiefly, perhaps not at all; its force will be spent mainly on that portion of the nervous apparatus which performs the function of translating to the mind the perceptions of sensitive nerves at the periphery; but it is not impossible that even the primary morbid action is occasionally developed in nervous centres which govern secretion and other functions of organic life; and that the dyspepsia, and other functional disorders of viscera, may in these cases be the *direct* result of a central disease, instead of reflex phenomena dependent upon the condition of consciousness, as is probably the case in many instances. In the later stages of the malady there can be no doubt that the mental depression reacts with great force upon the machinery of organic life, disordering secretions and rhythmic motions very extensively.

The *pathology* of hypochondriasis, in the strict sense of the word, does not exist, for there are no anatomical or physiological facts upon which it can be based. Morbid anatomy has revealed absolutely nothing which in the slightest degree explains the occurrence of the disease; and the physiology of the symptoms is to the last degree obscure and uncertain in its interpretation. It is only in those cases which develop into true insanity, more especially those which pass into dementia, that the brain exhibits any notable changes; and these alterations, when they occur, are no proper part of hypochondriasis. It is neither impossible nor unlikely that the improved modern methods of examining the nervous centres, if they could be applied to the central ganglia of certain visceral nerves (and especially to the nucleus of the vagus), might detect appreciable changes even in the early stages of the disease. But the opportunities for carefully examining the nervous symptoms of patients in the early periods of hypochondriasis can rarely be obtained, and it is probable enough that the question as to the pre-occurrence or not of structural changes will never be thoroughly cleared up.

The *treatment* of hypochondriasis consists in the use of moral and constitutional remedies and of remedies for symptoms.

It is obvious that the first duty of the physician is to encourage the hypochondriac to forget his woes; but nothing is so difficult in practice, and that for the best of reasons. It is a fallacy to suppose that the sufferings of the patient are unreal; on the contrary, they are most vividly real, and it is impossible that he should forget them till they cease. Yet the mind has a reflex influence upon the bodily disorder, which may be as effective for good as for evil; and this fact may be taken advantage of. The key to the moral treatment is the breaking down of the patient's morbid self-concentration, and this object may be achieved to some extent in many cases by a change in the course of his daily life. The class of patients with whom this may be most readily carried out are those in whom the constitutional tendency to hypochondriasis is aggravated by the *ennui* of an idle life: for these, an active career or pursuit of almost any kind is an immense gain; only the new occupation should be one which forces them to mix with the world. The isolated activity of the student is no real diversion from the fancies of hypochondriasis, as the case of Dr. Johnson, and of many other famous intellectual workers, abundantly proves. It is needless to say that all actively depressing influences should be removed, such as immoderate venereal indulgence, of whatever kind, or alcoholic intemperance. On the other hand, the influence of new emotions which tend to lift the patient out of himself can scarcely fail to be beneficial; and it would be a real good fortune to a hypochondriac if he could fall in love in a natural and healthy manner, or if he could interest himself warmly in philanthropic schemes or other plans of public usefulness. And, above all, something like a police supervision should be exercised as regards his studies, in order that he may be rigorously kept from the perusal of medical or other books which might remind him of his miseries; for though we do not believe that these things can create hypochondriasis, yet they may certainly prevent its cure. It is well understood, however, that no good can be effected by simply laughing at his narrative of suffering, or bantering him on his fancifulness; on the contrary, it is necessary for the physician to be interested, and to believe in the reality of his painful sensations. If the patient once thinks that the doctor is taking pains to get at the secret of his troubles, he will be inclined to accept the first word of encouragement the latter throws out; and the reflected influence of reviving hope will be certain to assist recovery.

The *constitutional* treatment is to be directed towards improving the general nutrition; and the task here is partly that of aiding the primary process of digestion of food, and partly that of rendering more active the processes of decomposition and exchange in the tissues generally. The hypochondriac either has a deficient, a capricious, or a ravenous appetite, but in any case the primary function of digestion is almost always markedly impaired if the disease has lasted for any length of time; and when this depends on a want of tone mainly, or a condition of irritation of the stomach (such as is indicated by a coated tongue with a red or strawberry tip), the use of vegetable bitters and mineral acids will often do great good. Defective secondary assimilation, which will be especially indicated by the condition of the urine, is generally much benefited by the use of cod-liver oil for a rather prolonged period, if only the remedy can be tolerated by the stomach. In cases where the oil cannot be borne, cream, butter, or some other form of fat, will often agree, and may be made the first stage to inducing the stomach to retain the cod-liver oil. Nor is it by any means only in cases where there is general emaciation that the administration of fat does good; it is probable that the nutrition of the nervous tissues is directly improved by the treatment in many instances. The administration of iron is doubtless of great use to some anæmic patients, and sea-bathing frequently appears to exercise a very beneficial influence: but the first of these remedies is generally most efficacious when taken in the form of the chalybeate waters of some foreign spa; and there is good reason to doubt whether both mineral waters and sea-bathing do not owe most of their apparent power to the moral influences of travel and change of scene and mode of life. The more specific nervous tonics, such as strychnia, quinine, or phosphorus, seem to exercise but a doubtful and accidental influence.

The treatment of symptoms is a thing to be eschewed in hypochondriasis, with certain special exceptions. While, however, it is desirable to avoid concentrating the patient's attention on parts which are the apparent seat of mere morbid sensations, it is important to relieve him of the distress caused by real (though mere functional) disorders of the digestive system. Decided acidity of the stomach should be counteracted by the use of antidotes, of which none is more efficacious than magnesia ponderosa, in ten-grain doses thrice daily, or Brandish's solution of potash, ten minims three times a day, with gentian or cascarilla. The excessive or too long continued use of alkalies is, of course, to be avoided. The distressing flatulence, which is often one of the earliest, and also one of the most annoying symptoms, is greatly relieved by creosote (one drop in a pill twice or thrice a day) or the infusion of valerian. Alcoholic tinctures should be very cautiously employed, if at all; for there is a real danger of the patient coming to appreciate the comforting sensations given by the spirit so highly, that he gradually takes to drink; this is especially true in the case of hypochondriacal women, as it notoriously is in hysteria. We may add that it is particularly likely to occur in patients exhausted by masturbation or other venereal indulgence. The constipation, which is frequently so obstinate and troublesome, must be remedied, if it be anyhow possible, without the use of drugs; for it is most dangerous to stimulate the patient's love of self-doctoring in the direction of the habitual use of purgatives. The prescription of fruit, green vegetables, &c., as articles of daily food, is a far more desirable mode of accomplishing our object; and the habitual practice of active bodily exercise is a powerful aid to the same end.

The question of the *quantum et quale* of physical exercise which may be beneficial in hypochondriasis forms a fitting subject with which to conclude our remarks on treatment, since this is a remedy which directs itself alike to the moral, the constitutional, and the symptomatic condition of the hypochondriac. The only rule, however, which it is possible to lay down for our guidance in this matter, is the direction to employ physical exercise in such a manner and to such an amount as shall fully exercise the muscles without ever producing severe fatigue, and shall also be amusing to the patient. It is a very dangerous error to carry exercise to the fatigue point; a short continuance of such malpractice will usually suffice to produce a profound deterioration of the vigour of the nervous system, and an aggravation of the hypochondriacal fancies.

ANOREXIA NERVOSA.

(APEPSIA HYSTERICA, ANOREXIA HYSTERICA.)[1]

In an address on medicine, delivered at Oxford in the autumn of 1868,[2] I referred to a peculiar form of disease occurring mostly in young women, and characterised by extreme emaciation, and often referred to latent tubercle and mesenteric disease. I remarked that at present our diagnosis of this affection is negative, so far as determining any positive cause from which it springs; that it is mostly one of inference from our clinical knowledge of the liability of the pulmonary or abdominal organs to particular lesions, and by proving the absence of these lesions in the cases in question. The subjects of this affection are mostly of the female sex, and chiefly between the ages of sixteen and twenty-three. I have occasionally seen it in males at the same age.

To illustrate the disease I may give the details of two cases, as fair examples of the whole.

Miss A—, æt. 17, under the care of Mr. Kelson Wright, of the Clapham Road, was brought to me on January 17th, 1866. Her emaciation was very great (*vide* Fig. No. 1,[3] p. 306). It was stated that she had lost 33 lbs. in weight. She was then 5 st. 12 lbs.; height 5 ft. 5 in. Amenorrhœa for nearly a year. No cough. Respirations throughout chest everywhere normal. Heart-sounds normal. Resp. 12; pulse 56. No vomiting nor

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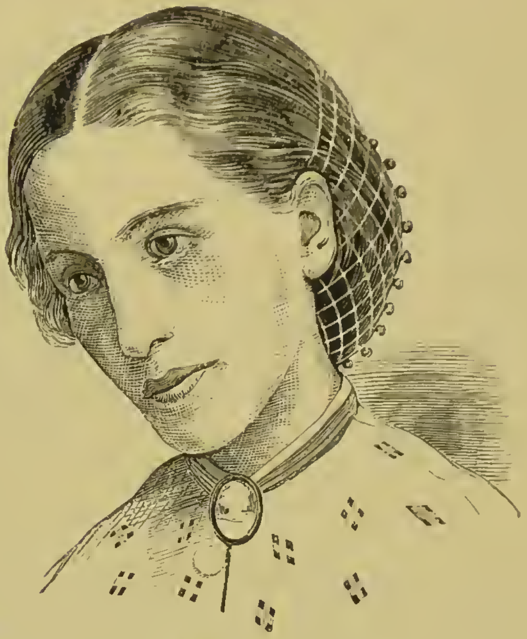
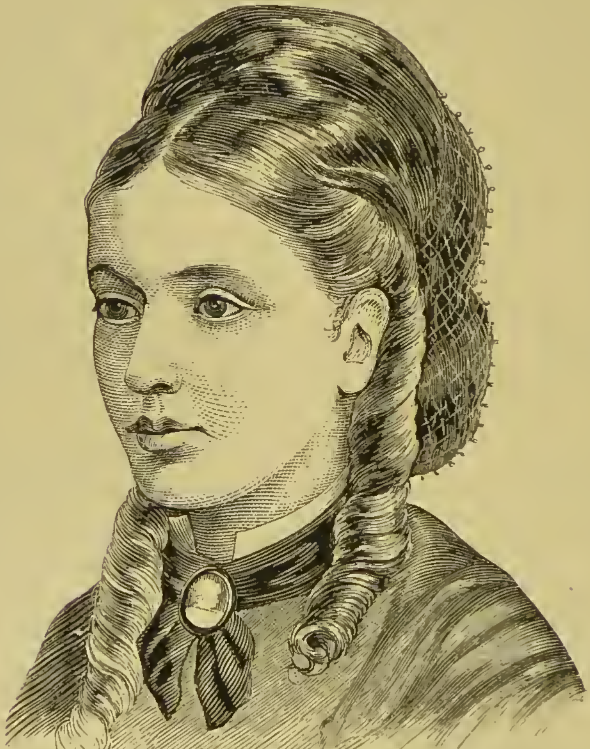
[1] Reprinted from the 'Clinical Society's Transactions,' vol. vii, 1874, p. 22.

[2] 'Lancet,' August, 1868.

[3] The woodcuts illustrating this paper are fac-similes of the original photographs exhibited at the time the paper was read.



Miss A—, No. 1.



Miss A—, No. 2.

diarrhœa. Slight constipation. Complete anorexia for animal food, and almost complete anorexia for everything else. Abdomen shrunk and flat, collapsed. No abnormal pulsations of aorta. Tongue clean. Urine normal. Slight deposit of phosphates on boiling. The condition was one of simple starvation. There was but slight variation in her condition, though observed at intervals of three or four months. The pulse was noted on these several occasions as 56 and 60. Resp. 12 to 15. The urine was always normal, but varied in sp. gr., and was sometimes as low as 1005. The case was regarded as one of simple anorexia.

Various remedies were prescribed—the preparations of cinchona, the bichloride of mercury, syrup of the iodide of iron, syrup of the phosphate of iron, citrate of quinine and iron, &c.,—but no perceptible effect followed their administration. The diet also was varied, but without any effect upon the appetite. Occasionally for a day or two the appetite was voracious, but this was very rare and exceptional. The patient complained of no pain, but was restless and active. This was, in fact, a striking expression of the nervous state, for it seemed hardly possible that a body so wasted could undergo the exercise which seemed agreeable. There was some peevishness of temper, and a feeling of jealousy. No account could be given of the exciting cause.

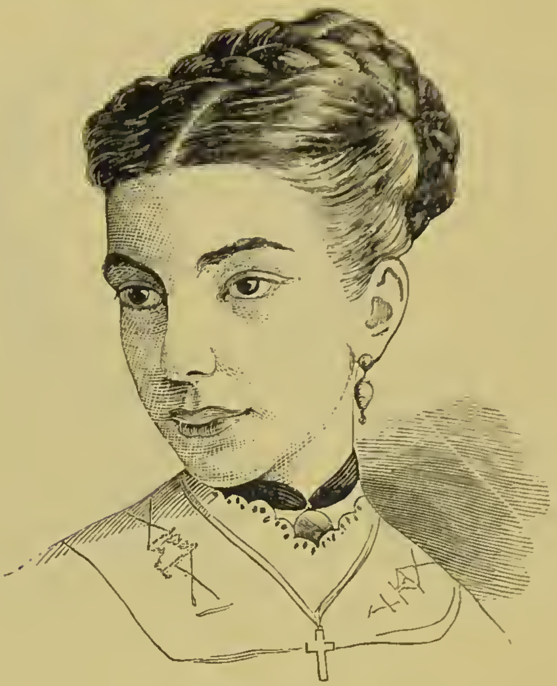
Miss A— remained under my observation from January, 1866, to March, 1868, when she had much improved, and gained in weight from 82 to 128 lbs. The improvement from this time continued, and I saw no more of her medically. The woodcut No. 2, p. 306, from photograph taken in 1870, shows her condition at that time. It will be noticeable that as she recovered she had a much younger look, corresponding indeed to her age, twenty-one; whilst the photographs, taken when she was seventeen, give her the appearance of being near thirty. Her health has continued good.

It will be observed that all the conditions in this case were negative, and may be explained by the anorexia which led to starvation, and a depression of all the vital functions, viz. amenorrhœa, slow pulse, slow breathing. In the stage of greatest emaciation one might have been pardoned for assuming that there was some organic lesion, but from the point of view indicated such an assumption would have been unnecessary.

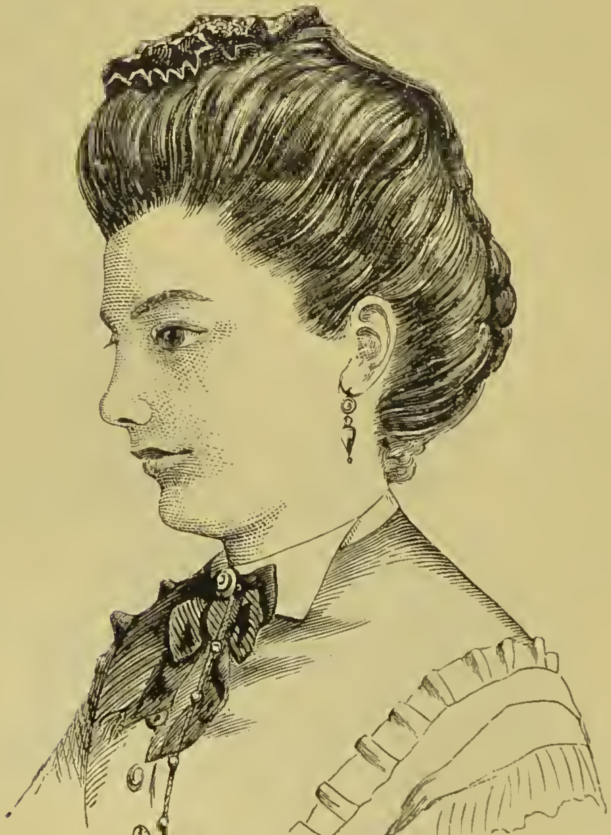
This view is supported by the satisfactory course of the case to entire recovery, and by the continuance of good health.

Miss B—, æt. 18, was brought to me, October 8th, 1868, as a case of latent tubercle. Her friends had been advised accordingly to take her for the coming winter to the south of Europe.

The extremely emaciated look (*vide* woodcut No. 1, p. 308), much greater indeed than occurs for the most part in tubercular cases where



Miss B—, No. 1.



Miss B—, No. 2.

patients are still going about, impressed me at once with the probability that I should find no visceral disease. Pulse 50, resp. 16. Physical examination of the chest and abdomen discovered nothing abnormal. All the viscera were apparently healthy. Notwithstanding the great emaciation and apparent weakness, there was a peculiar restlessness, difficult I was informed, to control. The mother added, "She is never tired." Amenorrhœa since Christmas, 1866. The clinical details of this case were, in fact, almost identical with the preceding one, even to the number of the pulse and respirations.

I find the following memoranda frequently entered in my note-book:—"Pulse 56, resp. 12; January, 1868, pulse 54, resp. 12; March, 1869, Pulse 54, resp. 12; March, 1870, pulse 50, resp. 12. But little change occurred in the case until 1872, when the respirations became 18 to 20, pulse 60.

After that date the recovery was progressive, and at length complete. (*Vide* woodcut No. 2, p. 308.)

The medical treatment probably need not be considered as contributing much to the recovery. It consisted, as in the former case, of various so-called tonics and a nourishing diet.

Although the two cases I have given have ended in recovery, my experience supplies one instance at least of a fatal termination to this malady. When the emaciation is at the extremest, œdema may supervene in the lower extremities, the patient may become sleepless, the pulse quick, and death be approached by symptoms of feeble febrile reaction. In one such case the post-mortem revealed no more than thrombosis of the femoral veins, which appeared to be coincident with the œdema of the lower limbs. Death apparently followed from the starvation alone. This is the clinical point to be borne in mind, and is, I believe, the proper guide to treatment. I have observed that in the extreme emaciation when the pulse and respiration are slow, the temperature is slightly below the normal standard. This fact, together with the observations made by Chossat on the effect of starvation on animals, and their inability to digest food in the state of inanition, without the aid of external heat, has direct clinical bearings; it being often necessary to supply external heat as well as food to patients. The best means of applying heat is to place an india-rubber tube, having a diameter of 2 inches and a length of 3 or 4 feet, filled with hot water, along the spine of the patient, as suggested by Dr. Newington of Ticehurst.

Food should be administered at intervals varying inversely with the exhaustion and emaciation. The inclination of the patient must be in no way consulted. In the earlier and less severe stages it is not unusual for the medical attendant to say, in reply to the anxious solicitude of the parents, "Let her do as she likes. Don't force food." Formerly I thought such advice admissible and proper, but larger experience has shown plainly the danger of allowing the starvation process to go on.

As regards prognosis, none of these cases, however exhausted, are really hopeless whilst life exists; and, for the most part, the prognosis may be considered favorable. The restless activity referred to is also to be controlled, but this is often difficult.

It is sometimes quite shocking to see the extreme exhaustion and emaciation of these patients brought for advice; yet, by warmth, and steady supplies of food and stimulants, the strength may be gradually resuscitated, and recovery completed.

After these remarks were penned, Dr. Francis Webb directed my attention to the paper of Dr. Laségue (Professor of Clinical Medicine in the Faculty of Medicine of Paris, and Physician to La Pitié Hospital) which was published in the 'Archives générales de Médecine,' April, 1873, and translated into the pages of the 'Medical Times,' September 6th and 27th, 1873.

It is plain that Dr. Laségue and I have the same malady in mind, though the forms of our illustrations are different. Dr. Laségue does not refer to my address at Oxford, and it is most likely he knew nothing of it. There is, therefore, the more value in his paper, as our observations have been made independently. We have both selected the same expression to characterise the malady.

In the address at Oxford I used the term *Apepsia hysterica*, but before seeing Dr. Laségue's paper, it had equally occurred to me that *Anorexia* would be more correct.

The want of appetite is, I believe, due to a morbid mental state. I have not observed in these cases any gastric disorder to which the want of appetite could be referred. I believe, therefore, that its origin is central and not peripheral. That mental states may destroy appetite is notorious, and it will be admitted that young women at the ages named are specially obnoxious to mental perversity. We might call the state hysterical without committing ourselves to the etymological value of the word, or maintaining that the subjects of it have the common symptoms of hysteria. I prefer, however, the more general term "nervosa," since the disease occurs in males as well as females, and is probably rather central than peripheral. The importance of discriminating such cases in practice is obvious; otherwise prognosis will be erroneous, and treatment misdirected.

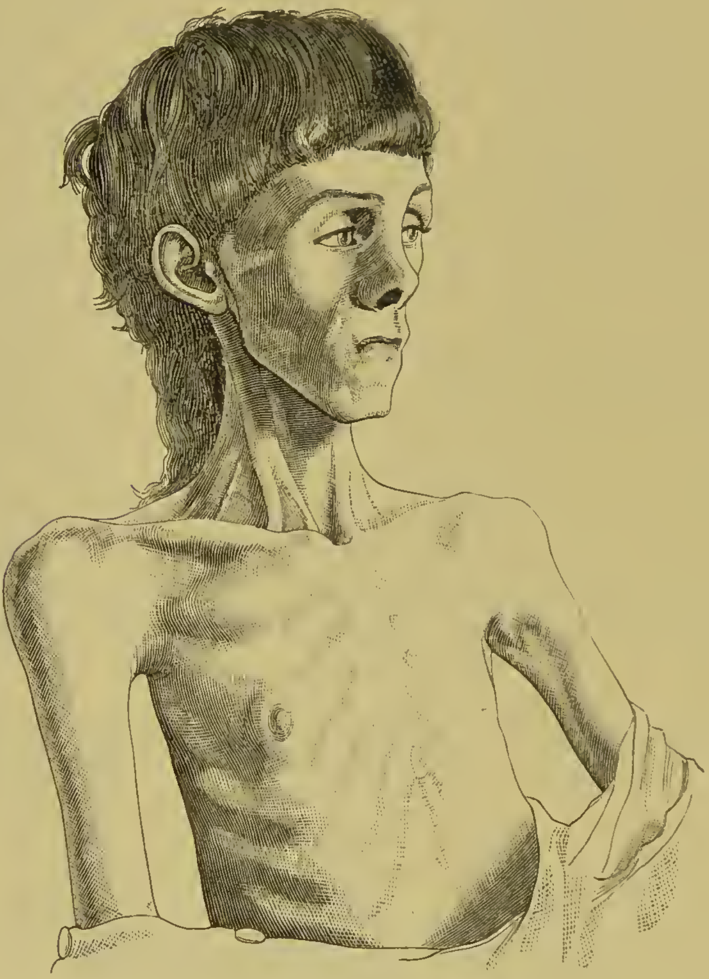
In one of the cases I have named the patient had been sent abroad for one or two winters, under the idea that there was a tubercular tendency. I have remarked above that these wilful patients are often allowed to drift their own way into a state of extreme exhaustion, when it might have been prevented by placing them under different moral conditions.

The treatment required is obviously that which is fitted for persons of unsound mind. The patients should be fed at regular intervals, and surrounded by persons who would have moral control over them; relations and friends being generally the worst attendants.

One other case is recorded as addendum to the above, in which the details are unimportant.

The following case was Sir William Gull's last contribution to the study of clinical medicine.

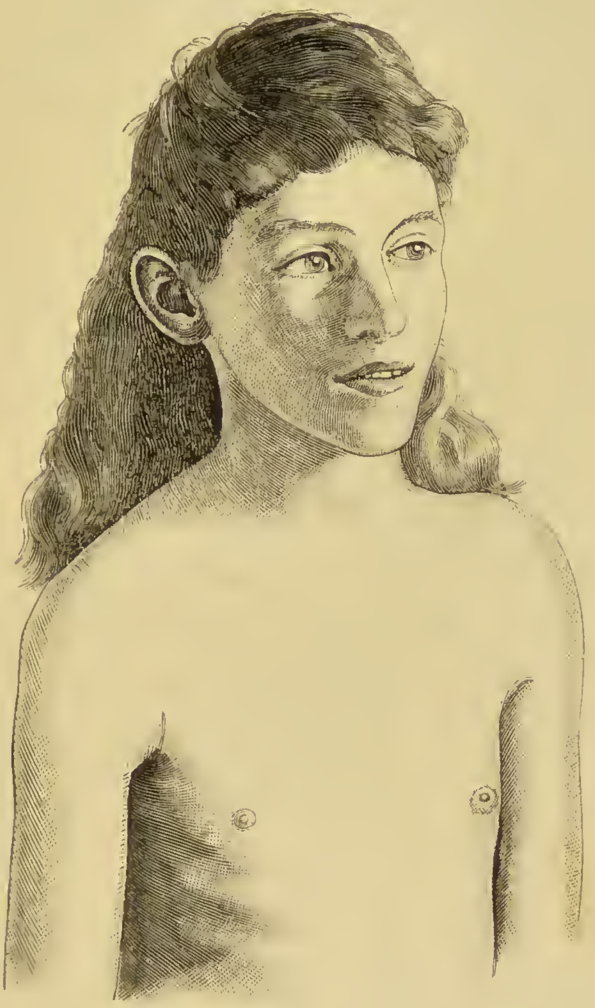
It may interest the readers of the 'Lancet' to look at the accompanying wood engravings, which were made from photographs of a case of extreme starvation (anorexia nervosa) which was brought to me on April 20th of last year by Dr. Leachman, of Petersfield. Dr. Leachman was good enough subsequently to send me notes of the patient's progress; and afterwards, at my request, the two photographs, taken by Mr. C. S. Ticehurst, of Petersfield. The case was so extreme that, had it not been photographed and accurately engraved, some assurance would have been necessary that the appearances were not exaggerated, or even caricatured, which they were not.



Photographed April 21st, 1887.

Miss K. R—, æt. 14, the third child in a family of six, one of whom died in infancy. Father died, aged sixty-eight, of pneumonic phthisis. Mother living and in good health. Has a sister the subject of various nervous symptoms, and a nephew epileptic. With these exceptions there have been no other neurotic cases on either side in the family, which is a large one. The patient, who was a plump, healthy girl until the beginning of last year

(1887), began early in February, without apparent cause, to evince a repugnance to food, and soon afterwards declined to take any whatever except half a cup of tea or coffee. On March 13th she travelled from the north of England, and visited me on April 20th. She was then extremely emaciated, and persisted in walking through the streets to my house, though an object of remark to the passers by. Extremities blue and cold. Examination showed no organic disease. Resp. 12 to 14; pulse 46; temp. 97°. Urine normal. Weight 4 st. 7 lbs.; height 5 ft. 4 in. Patient expressed herself as quite well. A nurse was obtained from Guy's, and light food ordered every



Photographed June 14th, 1887.[1]

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[1] This illustration differs from that given in the original paper; since, the negative having been destroyed, it was found impracticable to reproduce the woodcut.—Ed.

few hours. In six weeks Dr. Leachman reported her condition to be fairly good, and on July 27th the mother wrote, "K— is nearly well. I have no trouble now about her eating. Nurse has been away three weeks." This story, in fine, is an illustration of most of these cases, perversions of the "ego" being the cause and determining the course of the malady. As part of the pathological history, it is curious to note, as I did in my first paper, the persistent wish to be on the move, though the emaciation was so great and the nutritive functions at an extreme ebb.

ON A CRETINOID STATE SUPERVENING IN ADULT LIFE IN WOMEN.[1]

THE remarks I have to make upon the above morbid state are drawn from the observation of five cases. Of two of these I am able to give many details, but the three others were only seen by me on one or two occasions.

CASE 1.—Miss B—, after the cessation of the catamenial period, became insensibly more and more languid, with general increase of bulk. This change went on from year to year, her face altering from oval to round, much like the full moon at rising. With a complexion soft and fair, the skin presented a peculiarly smooth and fine texture, was almost porcellaneous in aspect, the cheeks tinted of a delicate rose purple, the cellular tissue under the eyes being loose and folded, and that under the jaws and in the neck becoming heavy, thickened, and folded. The lips large and of a rose purple, alæ nasi thick, cornea and pupil of the eye normal, but the distance between the eyes appearing disproportionately wide, and the rest of the nose depressed, giving the whole face a flattened broad character. The hair flaxen and soft, the whole expression of the face remarkably placid. The tongue broad and thick, voice guttural, and the pronunciation as if the tongue were too large for the mouth (cretinoid). The hands peculiarly broad and thick, spade-like, as if the whole textures were infiltrated. The integuments of the chest and abdomen loaded with subcutaneous fat. The upper and lower extremities also large and fat, with slight traces of œdema over the tibiæ, but this not distinct, and pitting doubtfully on pressure. Urine normal. Heart's action and sounds normal. Pulse 72; breathing 18.

Such is a general outline of the state to which I wish to call attention.

On the first aspect of such a case, without any previous experience of its peculiarity, one would expect to find some disease of the heart leading to venous obstruction, or a morbid state of the urine favouring œdema. But a further inquiry would show that neither condition was present; nor, when minutely studied, is the change in the body which I have described to be accounted for from either of these points of view.

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[1] Reprinted from the Clinical Society's 'Transactions,' vol. vii, 1874, p. 180.

Had one not proof that such a patient had been previously fine-featured, well-formed, and active, it would be natural to suppose that it was an original defect such as is common in mild cretinism. In the patient whose condition I have given above, there had been a distinct change in the mental state. The mind, which had previously been active and inquisitive, assumed a gentle placid indifference, corresponding to the muscular languor, but the intellect was unimpaired. Although there was no doubt large deposit of subcutaneous fat on the extremities, chest, and abdomen, the mere condition of corpulency, obesity, or fatness, would not in any way comprehend the entire pathology.

It is common to see patients with a very superabundant accumulation of fat in the subcutaneous adipose tissues, and on that ground more inactive, without the change in the texture of the skin, in the lips and nose, increased thickness of tongue and hands, &c., which I have enumerated. The change in the skin is remarkable. The texture being peculiarly smooth and fine, and the complexion fair, at a first hasty glance there might be supposed to be a general slight œdema of it, but this is not confirmed by a future examination; whilst the beautiful delicate rose-purple tint on the cheek is entirely different from what one sees in the bloated face of renal anasarca. This suspicion of renal disease failing, any one who should see a case for the first time might suppose that the heart was the faulty organ, and that this general change in the features and increase of bulk were owing to venous congestion. But neither would this be confirmed by an exact inquiry into the cardiac condition.

I am not able to give any explanation of the cause which leads to the state I have described. It is unassociated with any visceral disease, and having begun appears to continue uninfluenced by remedies.

CASE 2.—P. M—, æt. 40, a married woman, having had five children and living in good circumstances, came under my observation in 1866 complaining of general languor.

Heat was normal. Pulse 60. Catamenia too profuse. There had been gradual and general increase of bulk. The features had become broad and flattened, the skin was peculiarly fair and fine and soft, with a very delicate rose-bloom on the cheeks. The cellular tissue about the eyes was thrown into folds, giving the impression when cursorily looked at, of being œdematous. The eyes were bright, the lips were thickened, and of a light rose-purple. Tongue large, the speech guttural, and, as in the former case, as if the tongue were rather unwieldy. The sounds and impulse of the heart were normal, breathing was normal, urine normal. In fine, there was no discoverable change in any of the viscera, and the morbid state complained of seemed to be some primary change in the integuments, the muscles, and the nervous tissues of the cerebro-spinal system. This change continued to advance, so that in 1873 I made the following notes:

"Tongue large; false teeth cannot be worn, as tongue bitten by them. Lips large, thick, of a light rose (venous) tint. Features broad. Tissue under eyes loose, suggesting œdema. Fine delicate rose-tint on cheeks. Hair soft. Neck thick. Skin and subcutaneous textures lying in resisting folds. Hands broad and spade-like, the textures suggesting œdema, but not pitting. Much subcutaneous fat on chest, abdomen, and extremities. Thighs thirty-nine inches in circumference. Mind generally placid and lazy, but liable to being occasionally suddenly ruffled. Heart's action and breathing normal. Urine normal. Catamenia continue rather profuse."

The following is from a letter written by me on this case, March 7th, 1873, and fairly expresses my views of it at that time, which was seven years after my first observation of it.

"I believe it to be a rare form of constitutional disorder without any internal visceral disease, but characterised by great inaptitude to spontaneous exertion both of mind and body. The deposit of fat and the changes in the skin and connective tissues correspond to a languid condition of the venous circulation, but without any tendency to œdema, or any sign of cardiac defect.

"No doubt, under the stimulus of external circumstances, there is a response of mental activity which seems to prove that the mind requires but an exertion of the will to work up to its normal level. Though this be theoretically possible, I doubt if it be practically so in this state. The peculiar condition of the nervous system will, I believe, be best understood by reference to the external condition of the frame; for although I do not think the nervous centres have undergone any discoverable anatomical change, nor is there any evidence that the intellect is materially injured, I believe the nervous power is upon the whole lessened, and hence have arisen the changes in the temper, and the attacks which have been described to me.

"The best suggestions I can make are to let events take their course very much, maintaining the strength by simple regimen and fresh air, and by the occasional or more or less continuous use of such remedies as quicken the peripheral venous circulation—hot-air bath or warm bath, frictions, &c.; but the general good effect will, I think, be limited."

To those about such a patient the whole morbid condition is likely to be attributed to indolent habits, and the apparent incapacity for exertion to be deemed dependent upon mere inertness of the will. No doubt extreme circumstances have a distinct influence upon these as upon other patients, but I believe the disinclination to mental or muscular activity is largely pathological.

There is certainly a degree of habitual and mental indifference, though this may under occasional circumstances be obviated, since the intellect seems to be unimpaired. It will be noticed that I have designated this state *cretinoid*. My remarks are rather tentative than dogmatical, my hope being that once the attention of the profession is called to these cases, our clinical knowledge of them will in proportion improve. That the state is a substantive and definite one, no one will doubt who has had fair opportunity of observing it. And that it is allied to the cretin state would appear from the form of the features, the changes in the lips and tongue, the character of the hands, the alteration in the conditions of locomotion, and the peculiarities, though slight, of the mental state; for, although the mind may be clear and the intellect unimpaired, the temper is changed.

In an interesting paper[1] on *sporadic* cretinism occurring in England, my friend Dr. Fagge has given a case which began as late as the eighth year, in a subject previously healthy and well developed; and he states that in this case the physical configuration was alone manifested, or at any rate that any change in the mental powers was doubtful; and he adds "it may therefore be interesting to speculate as to what character would be present should the disease (if that be possible) arise still later in the course of adult life."

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[1] 'Medico-Chirurgical Transactions,' 1871, p. 155.

In the same paper we find that "in the report of the Sardinian Commission it is stated that, according to information received from medical men practising in infected districts, and according to all those who have written on this degeneration, there is no example in which, after the seventh year, a healthy child has become a cretin." And the Commission further quote with approval the statement of Maffei (who practised for a long time where cretinism was endemic, and who therefore had good opportunities of observing it), "that the period within which cretinism may commence is limited by the fourth year of life. . . It must, indeed, be mentioned that Rösch has recorded two cases in which the disease is said to have begun respectively at five years of age, and between seventeen and eighteen years."

It is to be borne in mind that these statements are applicable only to endemic cretinism, and therefore the objections from the experience of those who have observed only the endemic cases will be of less value.

The occasional occurrence of cretinism in children of healthy parents, and living in healthy districts in this country, is now well known. But our experience as to its development at different periods of childhood is of the most limited kind. The whole information on the point is contained, I believe, in Dr. Fagge's paper, and is illustrated by the second case given.

In the cretinoid condition in adults which I have seen, the thyroid was not enlarged; but from the general fulness of the cutaneous tissues, and from the folds of skin about the neck, I am not able to state what the exact condition of it was. The supra-clavicular masses of fat first described by Mr. Curling, and specially drawn attention to by Dr. Fagge as occurring in cases of sporadic cretinism in children, did not attract my attention in adults. The masses of supra-clavicular fat are not infrequent in the adult without any associated morbid change whatever.

On November 23rd, 1883, a discussion arose at the Clinical Society on a "typical case of Myxœdema" which was shown by Dr. Drewitt.[1] This debate elicited the following letter from Sir William Gull addressed to the 'Lancet.'

SPORADIC CRETINISM.

To the Editor of 'The Lancet.'

Sir,—I shall be obliged if you will find room for the following quotations from my late friend Dr. Hilton Fagge's paper on "Sporadic Cretinism." You will see from it that the sporadic cretinoid state has long been under consideration—at Guy's, at least,—and that such cases were brought forward in the clinical teaching there. The very great pathological value of Dr. Hilton Fagge's paper was his referring the state to atrophy of the thyroid, as will be seen in the last paragraph. We must all deeply regret that his very clear and highly informed mind should have been wanting to us in this interesting discussion.

Dr. Fagge writes as follows:—"The subject of sporadic cretinism appears hitherto to have attracted very little attention. I am not aware that anything has been written concerning it, with the exception of the papers which have already been quoted. In Virchow's great work on tumours I have failed to find any reference to it, either in the chapter on goitre (in which 'endemic cretinism' is discussed at some length) or in that on fatty tumours. It is, therefore, especially incumbent on me to state that, in the course of his clinical teaching at Guy's Hospital, Dr. Gull some years ago made me acquainted with many of the principal features exhibited by these cases. So far as I remember, the characters on which he laid most stress were the broad face, the flat nose and thick lips, the broad hands and feet, and the mild, tranquil disposition, so different from the mischievous tendencies of the idiots with whom these children are so generally associated. I do not think that Dr. Gull's attention had at that time been drawn to the presence of the peculiar tumours above the clavicles. He called the disease *cretinism*. . . . It is at this point, as I think, that the occurrence of sporadic cretinism, in association with an absence of the thyroid body, may be brought to bear upon the theory of the subject. We have but to suppose that the healthy thyroid body is capable of exerting such a counteracting influence, and that in most parts of England the cause of cretinism acts only with a low degree of power, and we can then at once see why a form of cretinism should show itself when the thyroid body is atrophied."—('Med.-Chir. Trans.,' 1871, vol. liv, pp. 166, 169.)

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[1] Clinical Society's 'Transactions,' vol. xvii, 1884, p. 49.

"Your obedient servant,

"WILLIAM W. GULL.

"BROOK STREET; *December*, 1883."

Note.—On December 14th, 1883, a committee was nominated by the President of the Clinical Society (with Dr. Ord as Chairman, and Dr. Hadden as Hon. Sec.) to investigate and report on "Myxœdema," the name which had been given by Dr. Ord to the disease called by Sir William Gull "the Cretinoid State."

This committee issued a "Report on Myxœdema" as a supplement to vol. xxi of the 'Clinical Society's Transactions,' 1888. This report contains a summary of 109 cases—ED.

SECTION II.

DISEASES OF THE VASCULAR SYSTEM.

ON THE

PATHOLOGY OF THE MORBID STATE

COMMONLY CALLED

CHRONIC BRIGHT'S DISEASE WITH CONTRACTED KIDNEY.

("ARTERIO-CAPILLARY FIBROSIS.")[1]

DR. BRIGHT and subsequent pathologists have fully recognised that the granular contracted kidney is usually associated with morbid changes in other organs of the body. The disease in the kidney and the co-existent morbid changes are commonly grouped together and collectively termed "chronic Bright's disease."

In this communication we propose to consider the pathology of this morbid condition. We are induced to do this because our observations tend to show that the present prevailing pathological theories do not fully comprehend the whole history of the disease.

It is, we believe, generally assumed that the kidney is the organ primarily affected, and in consequence a cachexia is induced through which other organs subsequently suffer and undergo chronic changes. How far this opinion is well founded we now proceed to examine.

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[1] By Sir William Gull, Bart., M.D., D.C.L., F.R.S., and Henry G. Sutton, M.B., F.R.C.P. Reprinted from the 'Medico-Chirurgical Transactions,' vol. lv, 1872, p. 273.

It will be advantageous first to consider the morbid changes in the kidneys, and subsequently the morbid changes which occur in other parts of the body.

The morbid anatomy of granular contraction of the kidney is generally so well known that it is needless here to fully describe it, though it is necessary to recount some of the histological changes which may be observed in this state.

For our examinations some sections were made of the diseased kidneys without any hardening process; others were made after freezing: some sections were faintly stained by a weak solution of nitrate of silver, others by carmine, and others were left in a natural state. The examination was made by $\frac{1}{5}$ and $\frac{1}{8}$ inch object-glass, and the following changes were noticed.

A fine fibroid or hyaline-fibroid substance was seen between the convoluted tubules, which made the tubules appear wider apart than normal.

In some parts this substance had a homogeneous appearance, in others it had a striated or fibroid appearance like a network, and contained minute indistinct nuclei (?), and in others it had a coarser and more defined fibre-like character. This substance was seen in considerable quantity round the Malpighian bodies, and in still greater amount in and around the walls of the minute arteries. In some renal arterioles injected with Beale's blue translucent fluid the elastic tissue of the tunica intima was normal. The muscular tissue appeared to be changed in some of the vessels—it seemed thicker than natural, particularly when the arterioles were viewed longitudinally. The muscular nuclei were indistinct, and many of them were so altered as hardly to be recognisable. External to the muscular nuclei there was a quantity of hyaline-fibroid substance, and the layer formed by this material was much thicker than the muscular layer (Plate VI, fig. 7). This hyaline-fibroid was in some of the arterioles bounded externally by a few coarser fibres; in some it merged imperceptibly into the fibroid tissues lying between the tubules. The arterioles were often very much thickened and tortuous. The lumen of some of them was encroached upon and narrowed, and in some it seemed completely obliterated. The morbid material here alluded to had in and around the walls of some of the arterioles very little hyaline appearance, but was more coarsely fibrous. Many of the convoluted tubules were not appreciably altered, except that their epithelium was more or less granular. Others were much shrunken and wasted. Here and there a quantity of the fibroid material was noticed, arranged in a concentric manner; and in the centre of the coil were a few indistinct, shrivelled, scarcely recognisable epithelial cells. These fibroid coils apparently enclosed atrophied tubules. Some of the tubules were irregularly dilated, or apparently formed a number of cysts. In some situations almost all trace of tubular structure had disappeared, and scarcely anything but fibroid tissue remained. When the cortex was very much contracted the Malpighian bodies were found lying very much closer together than normal, and surrounded by a considerable quantity of fibroid tissue (see Plate V, fig. 1).

In an early stage of granular contraction of kidney, when there are no changes appreciable to the naked eye, except that the surface of the kidney is, as technically called in the post-mortem room, "coarse," the following alterations may be observed. In the walls of some of the minute arteries, and also outside and around them, there appears to be an excess of fibroid tissue, and the arteries in consequence seem thicker than normal. The number of arterioles thus affected varies very much. Those most altered are in the cortical parts. In making this observation we are well aware that there is normally more or less of areolar tissue around the minute renal arteries, therefore it is difficult with any degree of certainty to appreciate any slight increase in such tissue; but after careful observation it seemed to us that the outer coats of the arterioles even at this early stage were thickened by increase of fibroid tissue. The muscular tissue did not appear increased. The intertubular tissue seemed also altered in some parts. It was abnormally distinct and clearly defined. The fibre-like appearance was unduly marked as contrasted with the normal kidney intertubular structure. Immediately under the capsule, and corresponding to the depressions of the granular surface, fibroid tissue extended inwards, obscuring and destroying the tubular structure. This appearance has been well described by Dr. Dickinson. Amongst this fibroid tissue we occasionally observed a number of corpuscles and nuclei.

It remains for us to describe the morbid changes in the epithelium of the convoluted tubules. Dr. Johnson (p. 219, first edition of his work on 'Kidney Diseases') states that in the early stage of the disease now under discussion the only deviation from the usual appearance is in the epithelial cells of the convoluted tubules. "The epithelial cells are," he says, "opaque, and have an unusual finely granular appearance: in some of the tubules there is an appearance as of entire cells having been shed filling the tubules and rendering them opaque; in others there is an equal filling and opacity of the tubules from contained epithelium in a disintegrated condition." Dr. Dickinson states, on the contrary, that in the early stage of granular renal disease the epithelium is as in healthy kidneys, but it is altered where the kidneys are greatly contracted. We also ourselves have observed in some specimens that the epithelium cells were finely granular, but natural in all other respects. In others we have noticed a quantity of granular or homogeneous matter that rendered the tubules more opaque, and almost completely concealed the epithelium. In the advanced stages of granular disease the cells were very irregular in shape and shrivelled; their nuclei were indistinct. In some spots the epithelial cells were absent, and in other tubules of the same specimen the epithelium was observed to be not appreciably altered.

In the kidneys which had recently been the seat of acute nephritis the epithelium was more opaque, and contained granules. Here and there only was an epithelium cell in its natural position, the rest of the tubule being denuded, or the cells concealed by granular matter.

From the descriptions given it would appear that Dr. Johnson, Dr. Dickinson, and ourselves have observed similar changes in the epithelium. From these appearances Dr. Johnson draws the conclusion that the secreting cells of the kidney have undergone a primary pathological change. He refers this change in the cells to their removing from the blood some poisonous materials with which it is charged, and so becoming themselves changed in appearance and structure. We submit that it is not necessary to adopt Dr. Johnson's explanation in order to account for these appearances and alterations in the epithelium. The granular appearance of the epithelium is by no means confined to Bright's disease. It is usual to find it in kidneys which present no sign of disease except merely mechanical venous congestion. This form of congestion is seen more or less in by far the majority of post-mortem examinations. It occurs after death from various causes, and it is certainly not dependent on any definite pathological state beyond that of venous obstruction, which notoriously always takes place to a greater or less extent during the process of dying:—in consequence of obstruction to the circulation through the right side of the heart the blood accumulates in the vena cava and in the renal and other veins; the renal venules and capillaries become distended, and serum with albumino-fibrinous contents transudes into the tubules, the urine becomes albuminous, and the epithelial cells are coated with granular fibrinous matter. This exudation may be moulded in the tubules, and form hyaline or other casts, which may be retained in the tubules or passed in the urine.[1] Kidney epithelium may also be shed as the epithelial cells of the bladder may soak off during or after death. Urine contained in the bladder is notoriously commonly albuminous after death, and charged with columnar and other epithelium. In such cases there is often no evidence of kidney or bladder disease. It appears to us, therefore, that Dr. Johnson has attached undue importance to these slight changes in the epithelial tissue, whilst the greater changes which are seen in the advanced stage of the granular renal disease may be regarded rather as a consequence than as a cause of the atrophy of the kidney tubules. Our observations, on the contrary, seem to show that the visible morbid changes in granular contracted kidneys are due to the primary formation of "fibroid" or "hyaline-fibroid" substance in the intertubular parts, including the vessels, and to atrophy of the tubular and intra-tubular structures of the kidney.[1]

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[1] Recent observations on the minute anatomy of the kidney raise a doubt whether casts formed in the convoluted tubercles of the cortex can escape from them.

This formation commences in different parts of the kidney commonly near the surface, but it also seems to us to commence in the outer coats of the arterioles and in the walls of the capillary vessels. From these parts it extends round the convoluted tubes and Malpighian bodies. This fibroid or hyaline-fibroid substance subsequently contracts and draws the Malpighian bodies together, compresses the urinary tubules and vessels, and may entirely obliterate them.[1] This thickening of the capillary walls and the diminished calibre of some of the arterioles must naturally interfere with the nutrition of the tissues, and tend to produce further atrophy. Owing to these changes in the capillaries and arterioles the quantity of blood passing to the secreting cells would be lessened, and in consequence diminished activity in the secreting function would occur and promote atrophy even in normal epithelium.

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[1] A criticism as to the method of preparation of specimens examined was made by Dr. G. Johnson, in reply to which the following letter was written to the 'British Medical Journal' (June 15th, 1872):—Ed.

"Arterio-capillary Fibrosis.

"Sir,—We think it our duty to refer to a short and extempore paper by Dr. George Johnson in your last issue, in which he makes some remarks upon arterio-capillary fibrosis.

"Dr. Johnson seems to draw conclusions, adverse to our observations, from the vessels of the pia mater of the sheep acted upon by acetic acid and glycerine. Dr. Johnson placed such a vessel before us, but without informing us previously what operation it had undergone; and it appeared to both of us that the tunica adventitia was abnormally thick. This admission seemed at the time to be eminently satisfactory to Dr. Johnson, as conclusively reducing to an absurdity all our previous observations.

"In reply to this, we desire to say no more than that we have no precise knowledge of the effect of the action of acetic acid and glycerine on the vessels of a sheep's pia mater. It is not a subject to which we have directed our attention. But we are able to say that the healthy vessels of a sheep's pia mater, not so acted upon by acetic acid (which reagent is well known to swell out the tunica adventitia), do not present the appearances simulating those described in our paper; and we now repeat, for the information of your readers, that in the investigations we have conducted neither acetic acid nor any other reagent which could distort the tissue was used. We may further add that both the diseased and healthy specimens were prepared and mounted by exactly the same methods, so as to form ground for strict comparison.

"A matter of so great importance as that in question cannot be decided by any *ex parte* evidence one way or the other. It demands, and will, we have no doubt, obtain a full investigation of competent judges, more unbiassed than we ourselves or Dr. Johnson can be. Why the vessels from certain subjects presented none of the changes described by us, whilst the vessels from others having renal and cardiac disease did present them, though both were prepared in the same way, we must leave Dr. Johnson to explain.

"We are, &c.

(Signed) "William W. Gull.
(Signed) "Henry G. Sutton."

We would not, however, maintain that the changes in the epithelium must be entirely secondary, for changes may be going on in it coincident with the hyaline-fibroid formation in the vessels.

Where the kidney disease was far advanced hyaline-fibroid changes were seen in the minute renal arteries precisely similar to those observed in the arterioles of the pia mater and of other parts of the body. Where the kidney disease was in an early stage the tissue around the arterioles of the kidney and between the tubules had a sharply defined, somewhat distinctive appearance. We have observed a similar appearance in the minute arteries of the pia mater.

Our examinations have shown us that this hyaline-fibroid formation commences, as regards the pia mater, in the outer coats of the arterioles and in the walls of the capillaries themselves; and observing that the same kind of formation occurs in the arterioles and capillaries of the kidney, we are led to infer that this change begins in the kidney as in the pia mater in the walls of the arterioles and capillaries, and subsequently extends to the surrounding structures.

We have now to consider the other pathological changes which make up the morbid condition known as chronic Bright's disease with contracted kidneys.

Of these the most important and, as we think, essential and primary, are the changes in the vascular system, more or less general throughout the body.

Dr. Bright and subsequent observers have recognised that arterial changes are part and parcel of this state.

Dr. George Johnson, in his work on 'Diseases of the

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[1] See Plate V, fig. 1.

Kidneys,' published in 1852, says that the walls of the minute renal arteries are usually much thickened, and that this is due to hypertrophy of their muscular layer.

Dr. Wilks, in the 'Guy's Hospital Reports' published in 1853, remarks that the occurrence of diseased arteries in the chronic form of Bright's disease is well known. It is necessary we should state that both he and Dr. Bright referred to atheromatous changes, and to the thickening which occurs in the larger and moderate-sized arteries—to changes in arteries visible, in fact, to the naked eye. Many other writers have made the same observations, but it is notorious that atheroma is common in the large vessels, not only in Bright's disease, but in other maladies; still there can be no doubt that it is exceedingly common in granular degeneration of the kidneys. Dr. Dickinson found it in the proportion of 52 per cent. Dr. George Johnson a few years ago showed that not only were the microscopical arteries of the kidneys thickened, but that the minute arteries of the skin and other parts of the body were similarly changed, and he attributed the change to hypertrophy of their muscular tissue.

Our observations confirm the opinion that the minute arteries are thickened in chronic Bright's disease, and we gladly acknowledge the debt the science of medicine owes to Dr. George Johnson in so distinctly insisting upon the fact.

Dr. Beale has also confirmed the accuracy of Dr. Johnson's observations as regards the thickening of the minute renal arteries, and he expressed an opinion that this is due not to hypertrophy of the muscular layer of the vessel; and he has stated that the outer layer of these vessels is thickened.

We have examined by aid of the microscope a large number of vessels taken from bodies in which there was more or less chronic disease of the kidneys, chiefly granular degeneration, and we now desire to bring the result of our observations under the consideration of the Society.

For the purposes of such examination we chiefly selected the vessels of the pia mater, since they offered the greatest facilities for our purpose, and most of the descriptions of the vessels and the measurements mentioned herein have therefore reference to the minute arteries of the pia mater. In a few specimens the arteries were injected, but the major part were in the natural state, or merely stained. The portions of pia mater were usually taken from the under surface of the cerebrum, where the membrane is thin. Afterwards the specimens were stained in carmine, and subsequently mounted in glycerine and camphor water. The vessels were then examined by $\frac{1}{5}$- or $\frac{1}{8}$-inch object-glass, and a first or second eye-piece. The outlines of some of the arterioles of each specimen were drawn by means of the camera lucida, and measured by a scale divided for the sake of convenience into $\frac{1}{20000}$ parts of an inch. This scale was adopted because it allowed of the estimation of minute differences.

It will be useful if we here mention that we generally found in the arterioles measuring about $\frac{1}{2000}$ part of an inch in diameter, that the lumen of the vessel was about twice as great as the thickness of its wall; *i. e.* on observing the vessel through the microscope we found the thickness of its two sides or walls was equal to the lumen.

In the larger arterioles the relative width of walls and lumen was different, the lumen being proportionately larger. In nearly all the cases from which the diseased vessels were taken the kidneys were more or less granular, and some much contracted. In a few cases only were the kidneys large, white, and mottled. The condition of the viscera is briefly given in the report of the cases, the details of which are recorded in the appendix to this paper.

The large arteries are usually not much thickened in chronic Bright's disease, but occasionally their outer layer is indurated and thickened by this fibroid substance. The greatest amount of thickening occurs in the coats of the arterioles and capillaries.

The outer layer of the arteries, measuring about $\frac{1}{1000}$ of an inch in diameter, are also not unfrequently thicker than natural. The degree in which the affected vessels are altered, and the extent to which the morbid change is diffused over the vascular system of the different organs, vary very much in different cases. In some, almost all the arterioles seemed more or less affected, in others only an arteriole here and there, the remaining vessels not being greatly if at all altered, though frequently their outline is more clearly and sharply defined than natural. A practised eye can usually recognise this early stage of change by this sharply defined outline. In consequence of the occasional limitation of the morbid change to a few arterioles, care is requisite in making an examination, or the diseased vessels may easily be overlooked.

The morbid change in an affected vessel is by no means necessarily uniform throughout its length, and may not extend to the branches it gives off. The lumen of the affected arterioles is sometimes distinctly lessened, and the diseased vessels are not unfrequently tortuous, whilst in the walls of some of the minuter ones it is common to find groups of fat-granules aggregated together. The morbid changes vary according to the size of the vessels affected. In the larger ones, in which the three coats are distinct, the inner layer (tunica intima) is sometimes thickened to a marked degree. When this happens the elastic tissue is seen to form the inner edge of the arterial wall, and outside this the tunica intima has a fine fibrous or molecular appearance.

The muscular coat is also variously altered. Thus where seemingly normal, if placed, as fresh as possible, in a strong solution of carmine for about twelve hours, the nuclei do not absorb the carmine so readily as in healthy vessels. This is a very distinct difference. The muscular layer seems also often relatively increased. This appearance is, however, to us equivocal, for with this apparent increase it is common to find, even in the same vessel, the muscular tissue wasted, and the nuclei irregular in shape, or reduced to small globular bodies, having the appearance of large fat-granules. The muscular coat may be, in fact, degenerated into granular matter at one part, whilst in a contiguous portion of the vessel it may seem to be relatively increased. The examination of this point which refers to the change in the muscular coat has much occupied our attention, but we have failed to discover evidence of the muscular hypertrophy so much insisted upon by Dr. George Johnson. Outside the muscular coat the morbid changes, to which we have already referred when describing the changes in the kidney, are well marked in the diseased vessels. The outer portion of the altered arterioles, as seen under the microscope, is commonly bounded by a few well-defined fibres of white fibrous tissue, within which, and immediately in contact with the muscular layer, there is a more or less homogeneous hyaline formation. Where this hyaline substance is in contact with the fibrous outer layer it has a fibrous appearance (see Pl. V, fig. 4). The general aspect of the affected vessels might give, as Dr. G. Johnson affirms, an appearance of true hypertrophy, but after full examination it seems to us the whole is due to a morbid process, and not to an increase of normal nutrition, this morbid process giving rise to the formation of the hyaline-fibroid substance we have described. In the otherwise homogeneous hyaline substance, ill-defined nuclei or corpuscles are often seen. These corpuscles cannot, when fully examined, be mistaken for normal muscle nuclei, since they are irregularly disposed in the tissue, and wanting in that definition characteristic of normal elements. Although in the more characteristically affected vessels the "hyaline" substance is, as stated, more or less homogeneous, still this is by no means uniform, for not only may it contain the bodies just referred to, but it may itself be more or less replaced by coarse fibroid and granular material.

The change from the "hyaline" to the "fibroid" character is probably due to slowness of formation, as it is more common in aged persons. It is to be stated that the fibroid changes do not occur in the smallest of the capillary arterioles. In these the homogeneous or nearly homogeneous "hyaline" change alone occurs.

The morbid appearances here described were chiefly studied in the minute arteries and larger capillaries of the pia mater, but, as we have said, the same may be seen in the arterioles of the kidneys, and, we may now add, of the skin, of the stomach, of the spleen, lungs, heart, retina, &c.[1]

In the arterioles of the spleen and lungs the morbid substance was more coarsely fibroid, and the pure hyaline change less distinct.

In reference to the seat of the hyaline-fibroid formation, we cannot pass over the question which will probably be raised by those histologists who believe in the existence of perivascular sheaths of the minuter vessels. On the existence of such canals we express no opinion, but assert only that the morbid changes we have noticed occur chiefly outside the muscular layer.

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[1] Plate V, fig. 2; Plate VI, fig. 1.

The following is a general summary of our microscopical observations:

1. That the arterioles throughout the body in that condition usually called chronic Bright's disease with contracted kidney, are more or less altered.
2. That this alteration is due to a "hyaline-fibroid" formation in the walls of the minute arteries, and a "hyaline-granular" change in the corresponding capillaries (see Pl. VI, figs. 1, 2).
3. That this change occurs chiefly outside the muscular layer, but also in the tunica intima of some arterioles.
4. That the degree in which the affected vessels are altered, and the extent to which the morbid change is diffused over the vascular system of the different organs, vary much in different cases.
5. That the muscular layer of the affected vessels is often atrophied in a variable degree (see Pl. VI, fig. 3).

In order to ascertain the extent to which this change in the vessels existed, the pia mater was examined in a large number of cases of persons who had died of various diseases. Fifty-five cases were thus examined, and the result is found in the appendix given, and shows that this "hyaline-fibroid" change was associated with granular contraction of the kidneys in most of the cases; but, in some few, the vessels of the pia mater had largely undergone this change; and the heart was hypertrophied, whilst the kidneys remained healthy.[1]

These examinations also showed that, in some cases, a few only of the minute arteries and capillaries were thickened, the left ventricle of the heart slightly dilated, whilst the kidneys were a little contracted in some of these cases, but in others not.[2]

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[1] See Appendix, Cases 2 and 3.

[2] See cases in the Appendix, Nos. 26 to 35.

We have further to state, that this "hyaline-fibroid" change was not found in the vessels of healthy persons who had been accidentally killed, nor in ordinary phthisis, nor in other diseases whose morbid conditions are not allied to the cachexia of so-called chronic Bright's disease with contracted kidneys.[1]

It is now to be asked, What is the morbid condition antecedent to the changes in the minute arteries and capillaries? Dr. George Johnson considers that an impure state of the blood induces this vascular change. In order that we may not do his opinion any injustice, it will be well to quote his own words. He says, "In consequence of the degeneration of the kidney, the blood is morbidly changed. It contains urinary excreta, and it is deficient of some of its own normal constituents. It is, therefore, more or less unsuited to nourish the tissues, more or less noxious to them. The minute arteries throughout the body resist the passage of this abnormal blood. The left ventricle, therefore, makes an increased effort to drive on the blood. The result of this antagonism of forces is that the muscular walls of the arteries and those of the left ventricle of the heart become simultaneously and in an equal degree hypertrophied. The persistent over-action of the muscular tissues, both cardiac and arterial, is registered after death in a conspicuous and unmistakable hypertrophy."

This theory does not appear to us supported by the facts.

Dr. George Johnson states that the change in the minute arteries is simply hypertrophy of the muscular coat. But our examinations show that they are thickened by a "hyaline-fibroid formation," and that in fact the muscular coat is often variously atrophied.

Thus Dr. George Johnson regards the changes in the heart and arterioles in chronic Bright's disease as a physiological result, due to a morbid change in the blood, whilst our observations lead us to regard the cardio-vascular changes as throughout a morbid one.

Further, extreme degeneration of the kidneys, and together with this, no doubt, a noxious state of the blood, to which Dr. Johnson attributes the vascular change, may be present, and the vessels may frequently be found healthy. In support of this statement we have recorded in the appendix cases of large white kidneys, and of scrofulous pyelitis,[1] in which the kidneys were much diseased, and the renal changes were chronic, attended with general œdema and uræmic symptoms, and the vessels were healthy. Dr. George Johnson and other pathologists regard, as we have said, the general vascular changes as essentially consecutive to antecedent renal disease, but our inquiries show that these changes are, or may be, independent of renal disease, and that the renal change in chronic Bright's disease with contracted kidney, when present, is but a part of a general morbid condition.

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[1] See particulars of cases given in the Appendix, Nos. 36 to 55.

We are led to conclude that the kidney disease does not give rise to the vascular change. Our investigations show the disease under the following forms:

(1) Kidneys often much contracted, heart much hypertrophied, minute arteries and capillaries proportionately thickened by "hyaline-fibroid" formation.[2]

(2) Kidneys little contracted, but heart much hypertrophied, minute arteries and capillaries much thickened by "hyaline-fibroid" substance.[3]

(3) Kidneys healthy, whilst heart much hypertrophied, and minute arteries and capillaries much thickened by "hyaline-fibroid" substance.[4]

These facts show that there is a morbid state in which the kidneys are contracted, the heart hypertrophied, and the minute arteries and capillaries altered by a "hyaline-fibroid" formation. Further that the kidney changes are often, but not always, a part and parcel of this morbid state. The absence of such lesions of the kidneys proves that they do not constitute an essential and indispensable part of the general process.

We have already mentioned that the granular contracted condition of the kidney is dependent on a "hyaline-fibroid" formation in its arteries and other structures. The same kind of morbid change, in fact, occurs in the contracted kidneys as occurs in the arteries and capillaries of the pia mater, of the skin, of the heart, of the stomach, and other parts. The kidney disease would, therefore, seem to be but a part, but not an invariable part of the "hyaline-fibroid" change; but as the vascular system was at some part affected with this change in all the cases, we regard such vascular change as the constant and essential part of this morbid state.

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[1] See Appendix, Cases 56 to 60.

[2] See Appendix, Cases 1, 4, 6, 8, 15. 21.

[3] See Appendix, Cases 7, 10. 20.

[4] See Appendix, Cases 2, 3, 19

We have next to consider the pathology of another morbid condition which forms part of the state known as chronic Bright's disease. We refer to hypertrophy of the left ventricle of the heart unaccompanied by any valvular defect or adhesion of the pericardium.

The morbid appearances of this hypertrophy are so well known that it is not requisite for us here to describe them, but it is necessary we should state that we have found the minute arteries in the walls of the heart much thickened by the formation of "hyaline-fibroid" substance already described.[1]

Bright, to account for this hypertrophy, says, "The most ready explanation appears to be that the quality of the blood is altered by the kidney disease. The blood in consequence affects the minute and capillary circulation so as to render greater action necessary to force it through the vascular system." Many pathologists have adopted this explanation. Dr. Wilks, however (see 'Guy's Hospital Reports' for the year 1853), has suggested that the hypertrophy may be dependent on atheromatous changes in the vessels.

They who adopt the explanation given by Bright, state in support of their opinion, that the hypertrophy of the heart and the renal disease are so frequently associated together as to show that there is some intimate relation between them. They further state that this hypertrophy occurs in all forms of chronic kidney disease; and therefore they conclude that it is consequent upon diminished excretory power of the kidney.

Dr. Johnson, who supports this opinion, further states that physiologists have demonstrated that impure blood circulates with great difficulty and creates an impediment.

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[1] Plate VI, fig. 6.

There is, however, evidence on the other hand which appears to be strongly opposed to these views.

Thus the cardiac hypertrophy and the renal disease are no doubt frequently associated, as we have said; but this does not prove that there is a relation of cause and effect between the two states, for it is evident that both these morbid conditions may be dependent on a third more general one. Moreover, against this commonly accepted explanation it can be shown that in many cases of chronic kidney disease the heart is not hypertrophied. Dr. Wilks has mentioned to us that in many cases of large white kidney he has found the heart free from hypertrophy. Dr. Dickinson states that simple hypertrophy of the left ventricle is rarely associated with any form of renal disease excepting granular degeneration.

We have post-mortem records of seventeen cases of large white kidneys, and in twelve of them the heart was healthy. Dr. Grainger Stewart found in lardaceous disease of the kidney that the heart was hypertrophied in only 4 per cent. of the cases. In four cases, in which the kidneys were almost destroyed by scrofulous pyelitis, we found the heart healthy.

We have particulars of nine cases in which the kidneys were very contracted and the heart was free from hypertrophy.

In these various forms of kidney disease the morbid changes were chronic, and it must be supposed that the blood was altered. The morbid conditions to which the hypertrophy is attributed were, therefore, present, but the heart, at the same time remaining healthy, shows that these renal and blood changes are not sufficient to produce hypertrophy, and indicates that when present it is due to some other condition.

In some of our cases, it is true, the kidneys were large, white, and mottled, and the heart was hypertrophied, but besides the renal affection the vessels in these cases were much diseased by the "hyaline-fibroid" formation.

We attribute the hypertrophy to the vascular change.

Dr. George Johnson explains the occasional absence of hypertrophy in cases of chronic Bright's disease by assuming that the muscular tissue of the heart is imperfectly nourished in such cases. In considering this suggestion it is necessary to bear in mind that if the blood be altered, and the circulation in consequence impeded, the left ventricle of the heart must contract with greater force than natural to overcome the impediment. To accomplish this it must acquire increased strength—become hypertrophied; or it would be unable to overcome the obstruction, and the ventricle then, being unable to empty itself completely, would in consequence become dilated.

Dr. Johnson may in this manner explain disproportionate dilatation, but does not explain how a normal-sized heart acquires the additional force requisite to overcome the supposed obstruction.

Experience has fully shown that hypertrophy of the left ventricle occurs much more frequently with granular contracted kidneys than with any other form of renal disease; and our observations, supported by the particulars given, appear to show that in this form of kidney disease the hypertrophy is not induced by the renal affection, but by a morbid change in the minute arteries and capillaries. Of thirty-four cases in which the kidneys were healthy, excepting that they were slightly granular, or in other words "coarse," and contained some cysts, the left ventricle was hypertrophied in eighteen; and there was no valvular disease or pericardial adhesion to account for the hypertrophy. In these eighteen cases the hypertrophy would appear to have been the older and preceding condition.

Niemeyer states[1] that Bamberger, arguing against the view that cardiac hypertrophy is dependent upon the renal disease, has shown that it begins in the earlier stages of Bright's disease. Observing, therefore, that this cardiac hypertrophy occurs not unfrequently in the very early period of the kidney affection, when the excretory function is not greatly altered; and further observing that the heart in some cases is not hypertrophied, when the kidneys are very much contracted, and the function of excretion is of necessity greatly altered, we also again conclude that the hypertrophy is not dependent on the kidney change.

The following particulars seem, in fine, to indicate that the

[1] 'Text-book of Practical Medicine,' vol. i, p. 300.

hypertrophy is induced, as we believe, by the morbid changes in the vessels.

The heart was found (see cases in the Appendix) hypertrophied in all the cases in which the vessels were much and generally thickened by the "hyaline-fibroid" change: the heart was found slightly hypertrophied where the vessels were a little thickened, or a few of them only were thickened by this "hyaline-fibroid" material; the heart was much hypertrophied when the vessels were much thickened, and there was no kidney or other disease, except this vascular change, adequate to account for it.

Nor is it difficult to explain how the vascular disease gives rise to the cardiac hypertrophy.

The "hyaline-fibroid" material in the walls of the arterioles must be an impediment to elasticity, and it can be experimentally shown that greater force is required to propel a fluid continuously through a non-elastic than through an elastic tube. The left ventricle, therefore, owing to this diminished elasticity of the arterial walls, has of necessity to contract with greater force to carry on the circulation.

It remains to briefly notice a few other morbid conditions which form part of the pathological changes known as chronic Bright's disease with contracted kidneys.

The first is vesicular emphysema. It is well known that vesicular emphysema and granular contracted kidneys frequently co-exist. Of thirty-three cases of persons about middle age in which the lungs were emphysematous, the kidneys were more or less granular, and contracted in twenty-two; and there were no changes in the lungs to show that such emphysema was compensatory. In some of the cases the emphysema was great whilst the kidneys were only slightly granular; in other cases the kidneys were much contracted whilst the lungs were comparatively little diseased, which facts seemingly show that the emphysema may precede the kidney contraction, or the kidney disease may occur antecedent to the lung disease; and this is fully borne out by clinical experience. We have not as yet had an opportunity of examining microscopically the vessels of a number of emphysematous lungs associated with contracted kidneys, but in a few such lungs we have found the vessels surrounded by what seemed to be an unusual quantity of fibroid tissue, and it is common to see without the aid of the microscope the connective tissue increased around the vessels and tubes in these emphysematous lungs.

With granular contracted kidney we have also found the aorta and aortic valves much diseased and the seat of atheromatous and sometimes calcareous changes, giving rise to dilatation of the aorta, with or without aortic obstruction and regurgitation.

Another morbid condition to be noticed is so-called "retinitis albuminurica," which experience has shown to be frequently associated with granular contracted kidney. In this change of the retina there is sclerosis of the connective tissue. There is also sclerosis and fatty degeneration of the coats of the blood-vessels, and the vessels have been found greatly narrowed and even obliterated. These morbid changes in some cases lead to atrophy of the optic disc and of the retina.[1] The tunica adventitia of the larger retinal vessels is often considerably increased in thickness. Mr. Bader, in describing these diseased vessels, says,[2] "Sclerosis is seen in the coats of the blood-vessels, especially of the small arteries and capillaries of the retina and choroid. Their walls are thickened through a homogeneous, strongly reflecting, not quite transparent substance."

From the description given it would seem, therefore, that the morbid appearances observed in these retinal vessels were similar to those we have observed in the arterioles and capillaries of the pia mater, kidneys, and other parts.

The spleen has been found by us in instances of chronic Bright's disease diminished in size; in some cases weighing only two or three ounces, and in some no more than one ounce and a half: capsule thickened; on section substance tougher than natural, but the most noticeable alteration was the increased quantity of fibrous tissue. Under the microscope the vessels were seen surrounded by a much larger quantity of fibroid tissue than usual, and in the outer coats of some of the minute arteries "hyaline-fibroid" changes were obvious, and similar to such as occur in the arteries of the kidney and pia mater. In some cases where the kidneys were greatly contracted the spleen was very much affected in the manner described, but in some cases the spleen was much wasted, and the kidneys were little contracted; in other cases the spleen was very little wasted whilst the kidneys were very much contracted.

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[1] See Mr. Soelberg Wells' 'Treatise on Diseases of the Eye,' p. 358.

[2] See 'Guy's Hospital Reports,' 1866.

We also found in many instances of granular kidney disease that the convolutions of the brain were much wasted, whilst the minute arteries of the pia mater were thickened by "hyaline-fibroid" substance. In other cases of contracted kidneys, in which the brain was healthy, no "hyaline-fibroid" changes were found in the vessels of the pia mater. In a few cases the convolutions of the brain were wasted, and the vessels of the pia mater were thickened by this "hyaline-fibroid" change, whilst the kidney and heart were healthy, showing the independence of this change of heart or kidney.

Fibroid changes with atrophy have also been observed by Drs. Fenwick and Wilson Fox in the intertubular portions of the stomach in chronic Bright's disease. The arterioles of the stomach have been found thickened. We have also observed in chronic Bright's disease with contracted kidneys great fibroid thickening in the walls of the minute arteries of the stomach, and fibroid changes in the intertubular parts.

The clinical history of this morbid state enables us to get clearer views of its pathology. Old age is not an entity, but it is pre-eminently a condition or set of conditions predisposing to that state which is called chronic Bright's disease with contracted kidneys. To demonstrate the extent to which man at different periods of his life is exposed to granular degeneration of the kidney, we have collected from post-mortem registers particulars of 336 cases, death being due to various diseases. These cases were grouped according to the age. The following is the proportion of granular degeneration of the kidneys, and the proportion of granular to healthy kidneys in each decennial period of life.

From the age of	The total number of deaths from all causes.	The number of those cases in which the kidneys had undergone granular degeneration.	The proportion of granular to healthy kidneys in each decennial period of life.
10—20 years.........	44	1	1 granular kidney in 44 healthy ones.
20—30 „	69	2	1 ditto in 34 ditto.
30—40 „	84	10	1 ditto in 8·4 ditto.
40—50 „	82	31	1 ditto in 2·6 ditto.
50—60 „	39	28	1 ditto in 2·3 ditto.
60—70 „	13	12	1 ditto in 1·08 ditto.
70—80 „	5	4	1 ditto in 1·2 ditto.

It here appears that after the age of forty a large proportion of persons who die of various diseases have more or less granular contraction of the kidneys as shown on the surface of these organs; and it further appears that after forty years of life granular degeneration greatly increases as age advances.

Granular degeneration of the kidneys, therefore, belongs principally to the period of life at or over forty years of age.

Dr. Dickinson shows that of 308 persons with granular kidney only 75 died before forty years, and 233 after forty years of age. It is evident, therefore, that chronic Bright's disease with granular kidney is allied with the conditions of age, and experience shows that it is caused by all those influences which are recognised as tending to bring about senile changes, whether prematurely or not.

Children are occasionally the subject of granular contracted kidney, and there is given in the appendix to this paper the case of a girl, aged 9 years, in whom occurred granular and very contracted kidneys, hypertrophied heart, and very thick "hyaline-fibroid" arterioles.

We here mention that occasionally in young subjects not over twenty years of age the kidneys may undergo extreme contraction and degeneration, and be apparently strictly a local affection, and death may occur from the so-called uræmic poisoning without any signs of the cardio-vascular changes characteristic of chronic Bright's disease with contracted kidneys of a later age. These cases, we believe, have another causation.

It has been shown above, and it is notorious, that many organs are diseased in the morbid state known as chronic Bright's disease with contracted kidneys; namely, the kidneys themselves, the minute arteries and the capillaries, the heart, the lungs, the aorta, the brain, the retina, the spleen, the stomach, and the skin.

But these various parts and organs are not constantly affected in the same order, nor is there any constant proportion between them as to the morbid changes each may undergo. In some cases all these organs are much diseased, and more or less equally so; in other cases the morbid changes are confined to a few and isolated parts.

In some cases the changes seem to commence in the kidneys or in the heart, sometimes in the lungs or in the brain, or perhaps in other organs.

Clinical medicine, especially as followed in private practice, enables us often to predict and trace these changes onwards until the morbid formation is general. Thus, a patient may come under care for headache and other allied symptoms, in whom, at a given stage, the renal and cardiac functions may be normal; and as the case goes on, the urine first, or the heart first, or the breathing may first give signs of further lesion, until, as the malady progresses, that state called chronic Bright's disease with contracted kidney may be fully produced, as shown by the thickened heart, the pale watery urine, the shrunken skin, the troubled brain, and the dimmed sight.

Observing that the pathological change may commence in various parts of the body, it might readily be surmised that the symptomatic phenomena must be very different in different cases. Experience fully shows that the symptoms are very varied in chronic Bright's disease with contracted kidneys; and we would maintain that its prodromata and the prominent symptoms in its course vary with the organ which is primarily or predominantly diseased. But whether many or few organs are affected, the minute arteries and capillaries of the diseased parts have been found by us thickened by "hyaline-fibroid" formation. With this "hyaline-fibroid" formation in the arterioles there is an atrophy of the adjacent textures. This appears to be a characteristic of this morbid change in whatever organ it occurs.

It will follow from these facts that we cannot, as sometimes done, regard the functional disturbances which occur in many organs during the course of chronic Bright's disease with contracted kidneys as dependent on blood changes only or chiefly. For instance, pain in the head, discomfort after food, palpitation, dry skin, epistaxis, are we believe due, not so much to changes in the blood as to the changes we have spoken of in the tissues themselves.

The conclusions to which we have arrived may be briefly summed up as follows:

1. There is a diseased state characterised by hyaline-fibroid formation in the arterioles and capillaries.

2. This morbid change is attended with atrophy of the adjacent tissues.

3. It is probable that this morbid change commonly begins in the kidney, but there is evidence of its also beginning primarily in other organs.

4. The contraction and atrophy of the kidney are but part and parcel of the general morbid change.

5. The kidneys may be but little if at all affected, whilst the morbid change is far advanced in other organs.

6. This morbid change in the arterioles and capillaries is the primary and essential condition of the morbid state called chronic Bright's disease with contracted kidney.

7. The clinical history varies according to the organs primarily and chiefly affected.

8. In the present state of our knowledge we cannot refer the vascular changes to an antecedent change in the blood due to defective renal excretion.

9. The kidneys may undergo extreme degenerative changes without being attended by the cardio-vascular and other lesions characteristic of the condition known as chronic Bright's disease.

10. The morbid state under discussion is allied with the conditions of old age, and its area may be said hypothetically to correspond to the "area vasculosa."

11. The changes, though allied with senile alterations, are probably due to distinct causes not yet ascertained.

Should it be considered necessary to distinguish this morbid state by any special term, we venture to suggest for that purpose the name "arterio-capillary fibrosis."

APPENDIX.

Case 1.—William L—,[1] æt. 43, who died January 1st, 1871. The autopsy showed the kidneys were very granular and contracted. They were mottled on their surfaces, and in their cortical parts there was a quantity of yellowish-grey matter, similar to what is seen in acute nephritis. The left ventricle of the heart was dilated and its wall much hypertrophied. The heart weighed seventeen and a half ounces. There was no valvular disease. Many of the arterioles of the pia mater were much thickened, and the measurements were as follows:

The relative width of the vessels.		Of the channel.		Of the sides or walls of the vessel.
11	...	4	...	7
15	...	5	...	10
17	...	6	...	11
10	...	2	...	8
15	...	5	...	10
28	...	8	...	13
14	...	5	...	9
16	...	6	...	10
30	...	14	...	26

The muscular layers of the arterioles were not increased. The thickening was due to the outer layer of the vessel being increased in size. This part had a very fine fibroid appearance. In some parts it appeared to be made up of homogeneous-looking matter. Many of the arterioles were in this manner diseased.

Case 2.—Sarah S—, æt. 63, who died January 25th, 1871. Autopsy showed that the cause of death was rupture of left ventricle of heart. The heart weighed fifteen ounces, and its left ventricle was dilated and hypertrophied. The kidneys weighed fifteen ounces; their surface was almost smooth; there was no decided granular change; the cortical parts were not contracted; the kidney substance was red, and showed venous congestion only. Very many of the arterioles were much thickened, and this was caused by a quantity of hyaline-fibroid substance outside the muscular layer, and the tunica intima in some of the vessels also was thicker than natural.

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[1] Further details of this patient and the cases following may be seen in the post-mortem registers of the London Hospital.

The relative width of vessel.	Of channel.	Of walls.
13	6	10
10	3	8
16	4	12
12	3	9

The muscular nuclei were very imperfect in some of the arterioles. In some parts they were normal; in others they were replaced by large clear granules, or they were entirely absent.

Case 3.—James D—, æt. 77, died of peritonitis following strangulated hernia. The autopsy showed that the left ventricle of the heart was dilated and hypertrophied. Its valves were healthy excepting a little atheroma in the mitral. The kidneys were venously congested; otherwise they were healthy. The walls of many of the arterioles were much thickened, and this was due to a granular fibroid growth outside the muscular tissue. The outermost part of the wall of the arterioles had a distinct fibroid appearance. Within this there was a homogeneous finely granular substance. Lying amongst this granular material were numbers of short, imperfectly developed fibres and oat-shaped nuclei. In some arterioles the muscular layer was well defined.

Relative width of vessel.	Of channel.	Of walls.
24	8	16
12	4	8
22	8	14
21	9	12
29	12	17
38	18	20

And in some of the arterioles the nuclei were faintly stained with carmine; in many other parts the muscular nuclei could not be seen, or they were very irregular in shape. The tunica intima had a coarse fibroid appearance, and in other arterioles a granular appearance. In some of the arterioles it was thicker than natural. Many of the vessels were in this manner affected.

Case 4.—James R—, æt. 58, died of suppurative pericarditis apparently pyæmic, July 25th, 1871. The left ventricle of the heart was much hypertrophied, and the aortic and mitral valves were normal. The kidneys were small, their surfaces markedly granular, and they contained cysts.

Relative width of vessel.	Of channel.	Of walls.
20	8	12
28	12	16
22	8	14
25	10	15
40	20	20

Some of the arterioles were much thickened, others were not. The thickening was not so great as is observed in some cases. The outer and inner coats of the thickened arterioles contained a quantity of hyaline matter, and to this change the increased size of the arteriole wall was due. The muscular nuclei were in parts shrivelled and very much wasted. The most marked disease was external to the muscular layer. The walls of the thickened capillary arterioles had a homogeneous granular appearance. A number of fat-granules were aggregated in some vessels.

Case 5.—James S—, æt. 39, died suddenly, apparently in consequence of dilatation of the left ventricle, on 15th May, 1871. Heart weighed 1 lb. 14½ oz. The left ventricle was very much dilated and its wall hypertrophied. The left auricle was also much dilated. Valves were normal. The spleen was small. The kidneys were faintly granular and much puckered on their surfaces; on section they appeared congested, otherwise the cortical and medullary parts were natural. Some of the arterioles were much thickened, but this change was mostly seen in the very minute ones.

Relative width of vessel.		Of channel.		Of walls.
22	...	9	...	14
23	...	9	...	14
31	...	14	...	17
30	...	10	...	20
20	...	8	...	12
20	...	6	...	14
30	...	12	...	18
33	...	15	...	18

The layer outside the muscular nuclei was much thicker than natural, and it had a distinct hyaline-fibroid appearance. The muscular nuclei were very defined and normal in some parts of the arterial wall, and absent in others. The capillary arterioles were also thickened, and they had a fine granular appearance.

Case 6.—John H—, æt. about 45. He was brought into the London Hospital dead on May 20th, 1871. The left ventricle of the heart was dilated and hypertrophied. The aortic valves were calcareous and incompetent. The lungs were emphysematous. The spleen was small. The brain was wasted. The kidneys were moderate in size, and they were very granular, and contained numbers of cysts. The cortical parts were smaller than natural. Many of the arterioles of the pia mater were much thickened. External to their muscular nuclei there was a hyaline-fibroid-looking substance, which made the external coat much thicker than normal. Many of the large capillaries were also very much thickened, and their walls had a granular or homogeneous appearance. The tunica intima of some of the arterioles was also thicker than natural.

Relative width of vessel.		Of channel.		Of walls.
28	...	9	...	19
27	...	7	...	13
25	...	8	...	17
21	...	8	...	13
21	...	9	...	12

Relative width of vessel.		Of channel.		Of walls.
33	...	10	...	23
20	...	7	...	13
13	...	5	...	8
23	...	9	...	14
11	...	3	...	8
25	...	9	...	16
25	...	9	...	16
14	...	4	...	10
10	...	2	...	8
30	...	9	...	21

Case 7.—Edward L—, æt. 42, died of erysipelas secondary to a scalp wound. Autopsy December 28th, 1870. The lungs were very emphysematous, more or less, throughout, and there was capillary bronchitis. Many of the minute bronchial tubes were filled with pus. The left ventricle of the heart was dilated and somewhat hypertrophied, also the right ventricle. The spleen and liver were natural; the kidneys were faintly granular, otherwise healthy.

Many of the arterioles and capillaries of the pia mater were much thickened. In the coats of the arterioles external to the muscular nuclei a quantity of hyaline-fibroid substance was seen, and to this change the thickening was due.

The nuclei of the muscular tissues were very indistinct in some parts and absent in others. The walls of the capillaries were thickened by the formation of a granular or homogeneous substance.

The relative width of vessel.		Of its channel.		Of its walls.
7	...	2	...	5
10	...	3	...	7
9	...	3	...	6
11	...	3	...	8
13	...	3	...	10
12	...	5	...	7
34	...	12	...	22
32	...	12	...	20
44	...	18	...	26
42	...	16	...	26

Case 8.—Emma C—, æt. 9 years. The kidneys were very much smaller than natural, especially the left; one weighed 2 oz., the other 1½ oz. The kidneys in consequence of being greatly contracted were much out of shape; their cortical parts were much reduced in size; their surfaces were very irregular and puckered, not finely granular. The heart's left ventricle was dilated and hypertrophied; its valves were healthy. The spleen was small and tough. Some of the arterioles of the pia mater were very much thicker than natural, owing to the layer outside the muscular layer being much increased.

The relative width of vessel.		Of channel.		Of walls.
30	...	12	...	18
32	...	12	...	20
36	...	14	...	22
34	...	14	...	20
36	...	10	...	26
20	...	6	...	14
20	...	6	...	14
18	...	6	...	12
20	...	8	...	12
20	...	8	...	12
18	...	6	...	12
20	...	8	...	12
18	...	8	...	10
18	...	5	...	13
14	...	6	...	8
20	...	8	...	12

The layer external to the muscular nuclei was thicker than the tunica intima and tunica media taken together, and in the larger arterioles it had a fine fibroid or hyaline-fibroid appearance. The thickening was most marked in the capillary arterioles or larger capillaries, and this change was due to the formation of granular hyaline substance in the walls of these vessels. The arterioles of the kidneys were much thickened by fibroid changes.

Case 9.—Margaret B—, æt. 68, died of acute meningitis following iridectomy. The kidneys were granular and cystic, but not apparently contracted. The left ventricle of the heart was slightly dilated and slightly hypertrophied. Some of the arterioles of the pia mater were much thickened, and their outer coats were much increased in size. This portion of the vessel was thickened by the formation of a hyaline-fibroid substance.

The relative width of the vessels.		Of the channel.		Of the walls.
35	...	14	...	21
52	...	19	...	33
25	...	7	...	18
38	...	10	...	28
21	...	5	...	16
27	...	6	...	21
15	...	6	...	9
22	...	5	...	17
25	...	8	...	17

Many of the arterioles appeared to be healthy. The outer coats of the larger arterioles were thickened by a fine fibroid substance. The thickened capillary arterioles had a homogeneous appearance.

Case 10.—Thomas B—, æt. 69, died of senile gangrene, May 4th, 1871. The kidneys were granular and cystic, but not contracted. The heart weighed 16¾ oz., the left ventricle was dilated and its wall hypertrophied; there was no valvular disease. The lungs were emphysematous and the brain atrophied.

The relative width of the vessels.	Of the channel.	Of the wall.
29	12	17
30	14	16
21	9	12
28	16	12
22	8	14
32	13	19
38	16	22
30	10	12
20	6	14
42	20	20
26	8	18
24	8	16

The outer coats of the arterioles were much increased in size, and the vessels in consequence were thickened.

Almost all the arterioles of the pia mater were thickened more or less, but the amount of thickening varied very much in the different vessels: in some it was little, so that it did not much increase the diameter of the vessels; in others it was very great, and it was due to hyaline-fibroid changes in the walls of the arterioles external to their muscular coats.

The tunica intima of some of the arterioles was also increased in size. The walls of the minute capillary arterioles were very much thickened, and they had a molecular hyaline appearance. The muscular nuclei of the arterioles were stained in parts, and they appeared to be normal; in other parts they were wasted and indistinct. A number of fat-globules were aggregated together in the walls of many of the minute vessels.

Case 11.—Frederick B—, æt. 31. The kidneys weighed 10¾ oz.; they were markedly granular, and the cortex was somewhat wasted, and they contained several cysts. The left ventricle of heart was hypertrophied and dilated; its valves were healthy; its right side was also dilated. The spleen was large and dark.

Relative thickness of vessel.	Of channel.	Of walls.
23	7	16
30	8	22
18	6	12
25	8	17
29	7	22
21	6	15
26	9	17
28	9	19
16	6	11

The cerebral arteries were atheromatous, and the brain was wasted; many of the arterioles of the pia mater were much thickened by hyaline-fibroid substance outside the muscular nuclei.

CASE 12.—Sarah R—, æt. 32, died August 24th, 1871. The kidneys were granular and contracted. The post-mortem appearances have not been described in the register.

The relative width of vessels.	Of channel.	Of walls.
21	8	13
22	8	14
23	9	14
24	6	14
20	8	12
20	7	13
31	9	22
27	8	19
23	7	16
21	8	13

The arterioles were very much thickened, and their coats outside the muscular layer were much increased in size; some of the arterioles were apparently unaffected, but many were thickened. The layer outside the muscular nuclei of the thickened arterioles was seen to consist of a hyaline-fibroid substance, and the thickening was caused by this new material. In this new formation a number of minute nuclei were seen.

CASE 13.—James P—, æt. 49. The kidneys were not granular, but large and mottled, and their appearance indicated that they had been recently the seat of acute nephritis. The left ventricle of the heart was dilated. The liver was in the condition known as incipient cirrhosis.

Many of the minute arteries of the pia mater were much thickened, and the layer outside the muscular tissue was increased; it was much thicker than the middle and inner coat taken altogether, and this thickening was due to a hyaline-fibroid substance. In some of the arterioles the muscular nuclei were very indistinct, and they were replaced by fat-granules; in others they were very distinct. The arterioles were not all equally involved. Some of the arterioles of the left ventricle of the heart were much thickened by fibroid formation, also the arterioles of the kidneys and skin. In the capillary walls fat-granules were aggregated in greater numbers. The relative size of the different parts of the vessels are given below.

Relative width of vessel.	Of channel.	Of walls.
15	5	10
13	5	8
15	6	9
12	5	7
30	10	20
19	7	12
25	10	15

Relative width of vessel.	Of channel.	Of walls.
28	12	20
15	5	10
9	3	6

The outlines of nearly all the minute arteries were unusually clearly defined. The channels of the thickened arteries were not appreciably encroached upon.

CASE 14.—George T—, æt. 67, died of cerebral hæmorrhage. We regret that we could not obtain detailed particulars of this case.

Many of the arterioles were very much thickened outside the muscular layer by a hyaline-fibroid substance. Amongst this new material were a number of indistinct corpuscles or nuclei which at first sight looked like muscular tissue, but the muscular layer was observed to be entirely distinct from those imperfectly formed elementary bodies.

The relative width of vessel.	Of channel.	Of walls.
24	9	13
22	7	15
57	21	36
38	9	30
57	21	38

CASE 15.—Charlotte A—, æt. 55. The kidneys were coarse on the surface; they were also wasted. They weighed 7¾ oz. Heart weighed 1 lb. 1¾ oz.; its left ventricle was dilated and hypertrophied. Many of the arterioles of the pia mater were very much thickened, and the coat outside the muscular layer was much increased by hyaline-fibroid formation. The new tissues in some of the thickened vessels were coarsely fibroid. The muscular nuclei were much altered in some of the arterioles.

The relative width of the vessel.	Of the channel.	Of the walls.
36	16	20
32	12	20
28	11	17
36	16	20

CASE 16.—James B—, æt. 48. Cause of death was dilatation of the left ventricle. The kidneys weighed 12½ oz.; they were very granular, and contained a few cysts. The cortical parts were very little contracted. Liver capsule was thickened. Heart weighed 1 lb. 8 oz., and the left ventricle was much dilated and hypertrophied. Very many of the arterioles of the pia mater were very much thickened, and their outer coats were much increased by the formation of hyaline-fibroid substance. The tunica intima in some arterioles was also much increased in size by the formation of hyaline-fibroid substance, and the larger capillaries were thickened by homogeneous or granular material.

In some of the arterioles the new tissue was very distinctly fibroid, and the hyaline appearance was not well marked. The muscular nuclei were wasted in some parts of these vessels, and the muscular layer seemed thicker than natural in other parts.

The relative width of the vessels.	Of the channel.	Of the walls
32	11	22
33	8	22
33	8	25

Case 17.—A man, æt. 64, had a cyst in the brain; died in the Whitechapel Workhouse. The kidneys were markedly granular, but not much contracted. The left ventricle was hypertrophied and dilated. For these particulars we are indebted to Dr. Ilott, the Resident Medical Officer of the Whitechapel Workhouse.

The arterioles in the pia mater were very much thickened, and the coat outside the muscular layer of these vessels was greatly increased by fibroid changes. This condition was very well marked in many of the arterioles, and the muscular nuclei were very distinct and clearly defined in some, and a good deal wasted in others.

The relative width of vessels.	Of channel.	Of walls.
18	6	12
36	13	23
36	14	22
42	15	27
31	9	22
36	14	22
35	11	24
30	8	22
26	8	18

Case 18.—James B—, æt. 50, died of acute nephritis; the kidneys were large and their surfaces smooth; were very much congested and mottled; some grey material was scattered amongst the highly congested tissue. The cortical portions had a similar appearance, and they were increased in size. The kidneys had the morbid appearances commonly seen in an early stage of acute nephritis. Heart: the left ventricle was much dilated and hypertrophied, and its valves were healthy. Many of the arterioles of the pia mater, especially the smaller ones, were very much thickened, and their outer coats increased in size. Some of the vessels were not uniformly thickened, but in parts only. The muscular nuclei were in some of the arterioles very indistinct and in other parts absent; they were replaced by a granular substance; outside the muscular layer there was a quantity of hyaline-granular or hyaline-fibroid substance which caused the thickening. The walls of the larger capillaries were infiltrated with a similar hyaline-granular substance; many capillaries were in this manner diseased.

Relative width of vessel.	Of channel.	Of walls.
19	6	13
20	7	13
32	11	22

Relative width of vessel.	Of channel.	Of walls.
21	8	13
27	9	18
40	13	27

CASE 19.—Samuel C—, æt. 62, died suddenly in a comatose condition. The brain was considerably wasted. The cerebral arteries were very atheromatous. Spleen weighed 4 oz.; liver 2 lb. 11 oz. The kidneys weighed 8 oz., and with the exception of the capsule being adherent they were normal. These organs were very carefully examined by Dr. Hughlings Jackson and Dr. Sutton, and they both agreed that they presented no other signs of disease. The heart weighed 15 oz.; it was much increased in size, owing to dilatation and hypertrophy of the left ventricle. Its valves were healthy. Lungs were emphysematous. Many of the arterioles of the pia mater were very much thickened, the layer outside their muscular tissue was much increased in size. This was due to a fine granular transparent substance. This new material was seen between the muscular nuclei and the coarser fibres of the tunica adventitia. The larger capillaries were also much thickened, and their walls had a granular or homogeneous appearance.

Relative width of vessel.	Of channel.	Of walls.
40	16	24
42	14	28
42	14	28
60	28	32
54	24	30
54	22	32
50	20	30
28	10	18
30	10	20
30	12	18
34	14	20
40	16	24
40	16	24
26	8	18
28	12	16
30	10	20
32	12	20
22	8	14
22	8	14
24	8	16

CASE 20.—William H—, æt. 51. There was softening of the cerebellum. The kidneys weighed 11¾ oz. They were granular, and they contained the remains of the two embolic blocks, otherwise they were healthy. The heart weighed 1 lb. 9 oz. All its cavities were dilated and the ventricular walls much hypertrophied; some vegetations on the aortic valves.

Many arterioles were very much thickened, and the layer outside the muscular nuclei was much increased in size. In this part a quantity of hyaline-granular and finely marked fibroid substance was seen. The walls of some of the arterioles were unequally thickened. The tunica intima was also thicker than natural, owing to the formation of a fine fibroid tissue. The muscle of the arterioles was much wasted in some spots, and replaced by bodies which looked like large fat-granules. The larger capillaries were very much thickened, and their walls had a fine granular appearance.

The relative width of vessel.		Of channel.		Of walls.
24	...	8	...	16
28	...	10	...	18
36	...	16	...	20
32	...	12	...	20
72	...	18	...	54
72	...	16	...	56
20	...	8	...	12
20	...	8	...	12
24	...	8	...	14
38	...	16	...	22
40	...	16	...	24
44	...	18	...	26
44	...	16	...	28
32	...	12	...	20
30	...	12	...	20
30	...	10	...	20

CASE 21.—William S—, æt. 56, died on November 24th, 1871.

Autopsy showed that the heart was enlarged; it weighed 19 oz. The right ventricle was much dilated, and the left ventricle was also dilated and hypertrophied. The lungs were emphysematous. Liver was in an early condition of cirrhosis. Kidneys weighed 11 oz.; their surfaces were granular. One kidney was much contracted, the other rather larger than natural. There were indications also of recent acute nephritis.

Microscopical examination of the pia mater showed that many of the arterioles were healthy, excepting that their outline was much more sharply defined than natural. Some of the arterioles were much thickened, and this was due to the increased size of their outer coat. The new formation in this part had a very fine hyaline-fibroid appearance. Some of the larger capillaries were very much thickened, and their walls were granular and translucent. A quantity of fat-globules were aggregated together in the coats of the minute vessels.

Diameter of the vessel.		Of the channel.		Of the walls.
16	...	5	...	11
22	...	7	...	16
40	...	15	...	25
23	...	10	...	13
32	...	17	...	15
36	...	20	...	16
26	...	13	...	13

CASE 22, a patient under the care of Dr. Hughlings Jackson, died in the Epileptic Hospital in November, 1871.

We are indebted to Dr. Jackson for kindly affording us the opportunity of examining the pia mater and kidneys. The left ventricle of the heart was much hypertrophied. The kidneys were very granular, and their cortical parts were very much contracted.

Many of the arterioles of the pia mater and kidneys were very much thickened. The layer outside the muscular nuclei was much thicker than natural, and this was due to the formation of hyaline-fibroid substance. The muscular tissue was not appreciably increased, but it was wasted in parts, and in some spots the muscular nuclei were entirely absent. The outer layer of the larger vessels was also thickened by a similar fibroid change.

Diameter of the vessel.		Of the channel.		Of the walls.
32	...	12	...	20
28	...	11	...	17
22	...	8	...	14
26	...	9	...	17
35	...	14	...	19
48	...	25	...	23
52	...	25	...	27
33	...	10	...	23
27	...	10	...	17
24	...	9	...	15
22	...	8	...	14

CASE 23.—Edward S—, æt. 46. Autopsy February 22nd, 1872. The brain was healthy; lungs were œdematous. Heart weighed 1 lb. 15¾ oz.; its left ventricle was very much dilated, and its wall much hypertrophied; valves and orifices healthy. Right ventricle and auricle were also dilated. Liver and spleen presented no noticeable change. Kidneys—their surfaces were granular and mottled. A quantity of greyish material was seen amidst highly congested tissue. The appearances were such as are usually considered to indicate acute nephritis. The cortical parts were very much contracted, so that the tubes in part almost reached the surface of the kidney. The cortex also mottled like the surface. The arterioles of the pia mater were healthy. They were very carefully examined, and they presented none of the hyaline-fibroid changes seen in other cases, nor were there any indications of thickening. The arterioles of the skin, stomach, and kidneys, were greatly thickened by the formation of hyaline-fibroid material.

CASE 24.—John J—, æt. 37. Autopsy April 22nd, 1872. The lungs were congested and œdematous. There was evidence of recent pericarditis. The heart weighed 1 lb. 2½ oz.; its left ventricle was much hypertrophied; its valves and orifices were healthy. The right ventricle was also greatly dilated. The capsule of the liver was slightly thickened; this organ was otherwise healthy. Spleen—its capsule was also slightly thickened, but its substance apparently healthy. Stomach was healthy. Kidneys were very granular, mottled on their surface, and greyish material scattered here and there, the latter apparently the product of acute nephritis. The cortical parts were much contracted. The arterioles of the skin were very much thickened by fibroid material. This new formation was seen outside the muscular layer. The fibroid formation had a coarse fibre-like appearance. The arterioles of the retina were also thickened by the formation of a hyaline fibroid material.

Case 25.—Elizabeth L—, æt. 42. Autopsy April 2nd, 1872. A very large blood-clot was found in the left hemisphere, outside the optic thalamus. There was a small clot in the pons Varolii, and a cyst in the right hemisphere. The left ventricle of the heart was considerably hypertrophied. Heart's valves and orifices healthy. The lungs, liver, and spleen were natural. The kidneys were very granular, small, and their cortex was much contracted. Many of the minute arterioles were thickened, some of them very much so by hyaline-fibroid substance outside the muscular layers. In a great many of the arterioles and capillaries a quantity of fat-granules were aggregated together.

Besides the above cases, in which the arterioles were very much thickened, we examined ten other cases in which some of the arterioles of the pia mater were thickened, though to a much less extent. In these cases the morbid condition appeared to be in a much earlier stage, but the histological changes were similar in kind though less in extent to those observed in the very thick vessels.

Case 26.—Elizabeth R—, æt. 44. The kidneys weighed 8 oz.; they were slightly granular and contained cysts. The lungs were extremely emphysematous. The brain was atrophied. The heart weighed 10½ oz.; its left ventricle appeared to be slightly dilated, and there was atheromatous disease of the aortic valves. The outer layers of a few of the arterioles were thicker than natural owing to the formation of a hyaline-fibroid tissue, and this new formation was seen outside the muscular nuclei.

The width of the arterioles.		Of the channel.		Of the sides or walls.
30	...	18	...	12
30	...	19	...	11
45	...	28	...	15
22	...	10	...	12
A 25	...	10	...	15
B 22	...	9	...	14
24	...	16	...	8
14	...	8	...	6

Case 27.—John S—, æt. 74, died of broncho-pleuro-pneumonia. The lungs were emphysematous; the liver atrophied. The spleen was also atrophied;

it weighed only 4½ oz. The kidneys were slightly granular, and not contracted; they weighed 15 oz. The increased weight was apparently due to venous congestion. The heart weighed 15 oz. There was no valvular disease, and the left ventricle was dilated and somewhat hypertrophied. A few of the arterioles were markedly thickened.

The width of the arterioles.	Of the channel.	Of the sides or walls.
A 10	3	7
B 13	5	8
C 57	24	33
D 32	10	22
12	6	6
14	6	8
10	4	6

The increased size of the arterial walls was due to the formation of a hyaline-fibroid tissue in the outer coat of the vessels. There was no evidence of muscular hypertrophy.

Case 28.—A man, æt. 60, died in the London Hospital August 28th, 1871, of epithelioma of the œsophagus. The heart was moderately dilated. Some of the arterioles of the pia mater were thickened by the formation of fine fibroid-looking tissue in their outer coats. There was no muscular hypertrophy; the walls of some of these thickened vessels were very irregular in size. The measurements are given, and the letters A B C D E F indicate the thickened ones. By far the greater number of the arterioles were not thickened. A large number of fat-granules were aggregated in the walls of the minute vessels.

The width of the arterioles.	Of the channel.	Of the sides or walls.
A 31	11	20
B 32	11	23
C 26	7	19
D 25	15	20
E 20	8	12
F 28	10	18
35	18	17
19	18	11
19	18	11
40	21	19
22	12	10
30	14	16

Case 29.—James C—, æt. 74, died in the London Hospital with disease of the knee-joint November 25th, 1871. The autopsy showed that the heart's muscle had undergone fatty degeneration. The condition of its cavities is not mentioned in the post-mortem record. The lungs were emphysematous; the arteries were very atheromatous; the renal capsules were adherent, and the kidneys contained cysts; the outer coats of some of

the arterioles were thickened by the formation of some coarse-looking fibroid tissue. The measurements of the thickened ones were as follows:

Width of the arteriole.		Of the channel.		Of the sides or walls.
19	...	7	...	12
13	...	5	...	8
17	...	7	...	10

Case 30.—George G—, æt. 57, died of acute peritonitis following hernia. Autopsy July 12th, 1871. The spleen was atrophied; it weighed 3½ oz. only. Liver atrophied, and weighed 2 lbs. 11 oz.; the kidneys weighed 11 oz.; they were slightly granular, otherwise natural; the heart weighed 12 oz. A few of the arterioles were slightly thickened in their outer coats by the formation of a fine hyaline-fibroid tissue; by far the majority of the arterioles were not thickened. The measurements and letters A B show the proportions of the slightly thickened arterioles; the remaining measurements show no evidence of thickening.

Width of the arteriole.		Of its channel.		Of its sides or walls.
A 39	...	16	...	23
B 46	...	21	...	25
31	...	15	...	16
47	...	27	...	20
38	...	22	...	16
51	...	28	...	23
38	...	27	...	11
18	...	9	...	9
10	...	5	...	5
14	...	7	...	7
11	...	6	...	5

Case 31.—A man, aged about 45, died of acute pneumonia. The kidneys were healthy, and the heart weighed 14 oz. Its left ventricle was slightly dilated. None of the arterioles were greatly thickened, but the walls of some of them were more sharply defined than natural, and the outer layers of a few arterioles were somewhat thickened, and had a fine fibroid appearance. The muscular nuclei were indistinct in many of the vessels. The measurements given show the amount of thickening:

Width of the vessel.		Of its channel.		Of its sides or walls.
42	...	19	...	23
42	...	20	...	22

Case 32.—A female died after removal of cancer in the breast, January 20th, 1872. The kidneys were granular, but not markedly wasted. Pericardium was adherent. The left ventricle of the heart appeared somewhat dilated; its valves and orifices were healthy; its muscle was very fatty. The arterioles of the pia mater were very sharply defined, and their outer layers seemed thickened by the formation of a fine fibroid tissue.

Case 33.—Robert B— died December 2nd, 1871. The autopsy showed vesicular emphysema. The heart was normal. The kidneys were large and mottled; they presented the appearances seen in a somewhat early stage of acute Bright's disease. The vessels were for the most part healthy; the only noticeable change was that the outlines of the arterioles were more sharply and clearly defined than natural. Only one arteriole was found with its outer layer thicker than natural, and it had a fine hyaline-fibroid appearance, and only one capillary arteriole was observed much thickened; its wall had a granular homogeneous appearance. The measurements indicated by the letter A show the proportions of the thickened vessel; the remaining measurements do not indicate any thickening.

Width of the vessel.		Of its channel.		Of the sides or walls.
A 22	...	8	...	16
32	...	10	...	12
34	...	20	...	14
42	...	25	...	17
44	...	26	...	18

Case 34.—William W—, æt. 63, died of cerebral hæmorrhage November 11th, 1871. The heart weighed 13 oz.; its left ventricle was somewhat dilated. There was no valvular disease. The kidneys weighed 10½ oz., and they were healthy. The spleen was wasted. The outer layers of some of the arterioles were slightly thickened by the formation of a fine fibroid tissue. This was most distinctly seen outside the muscular nuclei. In no place were the arterioles greatly thickened. The measurements given show the thickening was not great.

Width of the arteriole.		Of the channel.		Of the sides or walls.
24	...	10	...	14
16	...	8	...	8
28	...	12	...	16
36	...	14	...	22

Case 35.—Chas. M—, æt. 49. Autopsy December 9th, 1871; cause of death contusion of brain, the result of injury. Heart weighed 14 oz. Vegetations were seen on the aortic valves. Liver was congested, and weighed 3 lbs. 2½ oz. Kidneys weighed 9 oz.; their surfaces were slightly granular and cystic; cortical substance small. Some of the arterioles were thickened in their outer layers by the formation of a fibroid substance. The measurements given show the degree of thickening.

Width of the vessel.		Of its channel.		Of its sides or walls.
46	...	16	...	30
37	...	18	...	19
72	...	26	...	56

CASES *in which the minute arteries and capillaries were healthy, but there were fat-granules, so called, in the walls of some of the vessels.*

No.	Reference in post-mortem register.	Initials.	Sex.	Age.	Post-mortem appearances of viscera.	Condition of arterioles.	Measurements of the arterioles, made from a scale divided into $\frac{1}{20000}$ of an inch.		
							Diameter of arterioles.	Of channel.	Of walls.
36	July 26th, 1871.	Sarah N—	F.	—	So-called catarrhal pneumonic phthisis, or broncho-pneumonic phthisis; lungs very much diseased; other viscera healthy, excepting intestines.	The muscular nuclei were very distinct and stained very well; in the minute arterioles they almost touched the outer edge of the tunica adventitia; not the least evidence of thickening.	$\frac{22}{20000}$	$\frac{11}{20000}$	$\frac{11}{20000}$
							14	8	6
							18	10	8
							29	13	16
							17	8	9
							33	21	12
							31	19	12
							36	22	14
							17	8	9
							13	7	6
							21	13	8
37	Sept. 19th, 1871.	Sarah S—	F.	51	Acute tuberculosis, capillary bronchitis; kidneys healthy, excepting tubercle in them; heart healthy.	The muscular nuclei were distinct, not well stained; in the minute arterioles they almost touched the outer edge of the tunica adventitia; and the vessels were not thickened.	34	16	18
							30	15	15
							34	15	19
							25	10	15
							36	18	18
							44	27	17
							26	14	12
							23	12	11
							19	10	9
38	Nov. 27th, 1871.	John B—	M.	22	Acute tuberculosis; tubercular meningitis; lung studded with tubercle cavity at one apex; liver and kidneys contained tubercle.	The arteriole appeared to be healthy; and the muscular nuclei appeared to extend, or almost, to the outer edge of the tunica adventitia; vessels were not thickened.	24	12	12
							22	12	12
							26	10	6
							20	12	8
							20	10	10
							18	10	8
							16	8	8
							—	—	—

	474 of p.-m. record for 1871.				contents had escaped into the peritoneal cavity and set up acute peritonitis; kidneys were faintly granular and slightly contracted; heart weighed eight ounces; muscular tissue, valves, and its cavities, were healthy; liver adherent to cyst, otherwise healthy; brain healthy.	a sheath apart from the tunica adventitia; the muscular nuclei in the arterioles almost touched the outer edge of the tunica adventitia, and they were very distinctly seen; a few aggregations of fat-granules were seen in the walls of the vessels.	20 20 18 20 22 20	12 12 10 12 10 10	8 8 8 8 12 10
40	Nov. 22nd. 1871; page 470 p.-m. record.	James F—	M.	22	Fractured cervical vertebræ; spinal cord crushed; lungs much congested; pericardium healthy; heart, liver, spleen, brain, kidneys natural.	The arterioles appeared to be healthy; the muscular nuclei almost touched the outer edge of the tunica adventitia; the latter layers in some of the vessels had a very distinct fibrous appearance; there was no evidence to show that the vessels were thickened.	28 28 26 22 26 32 28 30	14 16 12 14 14 16 14 16	14 12 14 9 12 16 14 14
41	Nov. 20th, 1871; page 454 p.-m. record.	Frederick S—	M.	26	Pericardium not adherent; heart weighed 1 lb. 3 oz.; left ventricle much dilated; aortic valves, when tested by pouring water in the aorta, were found to be incompetent; lungs were studded with apoplectic masses; liver slightly cirrhosed; spleen firm and congested; also intestines and kidneys were congested.	The arterioles appeared to be healthy; the muscular nuclei almost touched the outer edge of the wall of the vessel; in other parts there was a thin layer of tunica adventitia intervening; the muscular layer was everywhere very distinct; the tunica adventitia was not increased; there were a few fat-granules aggregated in the walls of the vessels.	30 32 32 30 32 30 36 36	16 20 18 18 18 14 24 22	14 12 14 12 14 16 12 14
42	Nov. 15th, 1871.	—	M.	—	Fractured pelvis; lacerated liver, spleen, kidneys, and diaphragm.	Arterioles appeared to be healthy; there were no indications of thickening; muscular nuclei distinct, and they almost reached the outer edge of the wall of the vessel.	18 18 28 32 32 42 44	12 12 20 18 20 24 26	6 6 8 14 12 18 18

No.	Reference in post-mortem register.	Initials.	Sex.	Age.	Post-mortem appearances of viscera.	Condition of arterioles.	Measurements of the arterioles, made from a scale divided into $\frac{1}{20000}$ of an inch.		
							Diameter of arterioles.	Of channel.	Of walls.
43	Sept. 21st, 1871.	William S—	M.	27	Caries of the right ilium; kidneys and heart were healthy.	The transverse muscular fibres of the arterioles had undergone fatty degeneration; they were studded with fat-granules; in other respects these vessels were healthy; no indication of thickening.	38	24	14
							39	20	19
							18	9	9
							50	29	21
							35	20	13
							34	16	18
							22	12	10
44	Oct. 12th, 1871.	Jane G—	F.	20	Gliomatous tumour in the cerebellum; the other viscera were healthy.	These arterioles appeared to be healthy, and there were no indications of thickening except in one capillary arteriole; the specimen was very carefully examined, and only one vessel was found in this condition; some fat-granules were aggregated in the walls of some of the vessels.	56	36	20
							29	15	14
45	March 30th, 1872.	George W—	M.	52	Autopsy, London Hospital; there was medullary cancer of stomach, colon, peritoneum, pleura, and of abdominal and thoracic glands; heart's muscle soft and apparently fatty, in other respects this organ was healthy; kidneys normal; liver contained cancer.	The arterioles of the pia mater were healthy; the muscular nuclei were well stained, and were lying close to the outer edge of the arteriole wall; no hyaline-fibroid changes.	—	—	—
							—	—	—
							—	—	—
							—	—	—
							—	—	—
							—	—	—
							—	—	—
							—	—	—
46	—	—	—	—	Fractured vertebræ; spinal cord crushed.	The arterioles of the pia mater were healthy; no indications of thickening; muscular nuclei were distinct; some of the arterioles were injected with Beale's blue injecting fluid.	66	44	22
							66	40	20
							52	30	20
							54	34	20
							48	26	22
							44	24	20
							42	24	18
							40	24	16
							48	26	[illegible]

					tricle natural, vegetations on aortic valves, the remaining valves and the orifices were natural; kidneys and brain were natural.	granules being aggregated here and there; no indications of thickening; no hyaline-fibroid change.	28 24 24 26 14 18	14 13 12 16 8 10	14 10 12 10 6 8
48	Nov. 18th, 1871.	David E—	M.	32	Death due to the administration of chloroform; viscera healthy; muscle of heart soft, and its left ventricle slightly dilated; heart weighed 14½ oz.	Arterioles presented no abnormal appearances; no indications of hyaline-fibroid changes.	22 38 26 19	12 17 14 11	10 15 12 8
49	March 7th, 1872.	—	—	17	Autopsy: viscera healthy; death following injury to right hand and arm.	Arterioles of pia mater were healthy; no indications of hyaline-fibroid changes.	— — —	— — —	— — —
50	March 5th, 1872.	William E—	M.	35	Death due to poisoning by corrosive sublimate; there was œdema of larynx and false membrane on the mucous surface of trachea; mucous surface of large intestine congested; rest of viscera healthy.	Arterioles of pia mater were healthy; their muscular nuclei were well stained, and lying near the outer edge of the arteriole wall; no hyaline-fibroid changes.	— — — — — —	— — — — — —	— — — — — —
51	Jan. 27th, 1872.	Ann C—	F.	25	Autopsy: death due to bronchitis; heart, right ventricle much hypertrophied and dilated; pericardium thickened; lungs bound down by old adhesions; remaining viscera healthy.	Arterioles of the pia mater were healthy, they presented no hyaline-fibroid appearances; muscular nuclei well stained.	— — — — — —	— — — — — —	— — — — — —
52	Nov. 21st, 1871; page 466 p.-m. record.	—	F.	51	Heart was small, and muscle pale and studded with yellow markings indicative of fatty degeneration, valves and orifices natural; liver capsule thickened; spleen normal; kidneys pale, otherwise they were normal; intestines and other viscera healthy.	The arterioles were apparently healthy; their outline was not sharply defined, and there was no evidence of thickening or of hyaline-fibroid change; the muscular nuclei almost touched the outer edge of the walls of the vessel.	24 22 36 38 36 36	12 12 22 22 22 20	12 10 14 16 14 16

No.	Reference in post-mortem register.	Initials.	Sex.	Age.	Post-mortem appearances of viscera.	Condition of arterioles.	Measurements of the arterioles, made from a scale divided into $\frac{1}{20000}$ of an inch.		
							Diameter of arterioles.	Of channel.	Of walls.
53	Feb. 21st, 1872.	—	M.	56	Autopsy showed acute endocarditis; left ventricle and muscle of heart dilated; disease of the aortic valves; acute pneumonia and pleurisy; morbid changes in the kidneys, indicating acute nephritis.	There were no indications of thickening in the vessels of the pia mater; the nuclei of the muscular fibres almost touched the outer edge of the arterioles, and there was in consequence very little appearance of tunica adventitia; there were no aggregations of fat-granules.	38 44 27 31	23 25 15 16	16 19 12 15
54	Jan. 24th, 1872.	—	F.	43	Autopsy showed broncho-pneumonic phthisis.	The arterioles were healthy; there was no evidence of thickening; the muscular nuclei were seen close to the outer edges of the walls of these vessels; there were no large aggregations of fat-granules.	29 44 16	17 28 9	12 16 7
55	Dec. 4th, 1871.	—	M.	62	Stricture of the urethra; fibroid degeneration in the lungs; dilated right ventricle of heart; liver cirrhosed; hypertrophied brain.	The arterioles were healthy; the muscular nuclei were very well stained, and they were seen lying close to the outer edge of these vessels; there were no aggregations of fat-granules.	50 42 40 40	28 23 22 22	22 19 18 18

The arterioles in some cases were free from thickening, and the heart was not hypertrophied; whilst there was chronic disease of the kidney, viz. large white kidney and so-called scrofulous pyelitis.

Case 56.—This occurred in a patient named William E—, æt. 33. He was in the London Hospital many weeks with great and general œdema, and the urine contained a large quantity of albumen. While in the hospital he had attacks of great dyspnœa, also of vomiting; and a week or two before he died he had convulsions alternating with coma.

The autopsy showed that the kidneys were larger and much heavier than natural. Their capsules separated readily; their surfaces were smooth and white, and very few vessels were seen. The kidneys had the peculiar, general, marble-white appearances so well represented by Dr. Bright in one of his drawings of Bright's disease. The cortical parts were much increased in size, and appear to be infiltrated with similar white material. Heart was normal. The arterioles of the pia mater were not thickened.

Case 57.—In the case of a man who died in the London Hospital, under the care of Dr. Andrew Clark, there was general œdema; the urine contained a large quantity of albumen, and there were symptoms of uræmic poisoning. The kidneys were in the morbid condition known as "large, white, and mottled." Heart was normal. The arterioles of the pia mater were carefully examined, and there were no indications of thickening.

Case 58.—In a third case, a girl æt. 7 years, who died in the London Hospital under the care of Dr. Down, February, 1872, there were large white kidneys and general œdema. Heart was natural. The arterioles were not thickened. The disease in this case was of considerable standing, for the patient had been under Dr. Down's observation with albuminuria for more than twelve months, and she was in the London Hospital with kidney disease on more than one occasion.

Case 59.—A fourth case was that of George G—, æt. 18. He was in the London Hospital several weeks with general œdema and a large quantity of albumen in the urine. He died with symptoms of uræmic poisoning. The autopsy on May 6th, 1872, showed the kidneys weighed 14¾ oz. They were much larger than natural, and their capsules separated readily; their surfaces were smooth and contained no cysts. They were of a pale fawn-colour, and a large number of yellow spots were seen scattered in the cortical parts. The cortex was greatly increased in size, and converted into similar fawn-coloured substance. These organs were good examples of large white kidneys in an advanced stage of disease. Heart weighed 8 oz.; there was no dilatation or hypertrophy; its valves and orifices were healthy. Liver, spleen, and lungs were healthy. The walls of the arterioles were not thickened. In the minuter ones the muscular nuclei were situated close to the outer edges of the vessel.

CASE 60.—A fifth case was that of William L—, æt. 27, who died in the London Hospital. It was stated that he had been under the care of Dr. Thorowgood, at the Victoria Park Hospital, for many months, with symptoms of phthisis and strumous pyelitis. The capsules of the kidneys were very much thickened and the right kidney was adherent by old tough fibrous bands to the liver and intestines. On section this kidney seemed almost completely destroyed. It contained several cavities filled with thick puriform matter. There was very little kidney structure remaining. The left was similarly affected, but not to the same great extent. Heart healthy.

The arteries of the pia mater were injected with Beale's Prussian blue solution, and afterwards carefully examined by aid of the microscope, and there were no indications of arterial thickening.

In other cases (see case No. 13, James P—, æt. 49) the kidneys were large, white, and mottled, the arterioles were thickened by hyaline-fibroid changes, and the left ventricle of the heart was dilated.

In the latter case we cannot conclude that the kidney disease induced the morbid changes in the arterioles and heart; for there is no evidence to show that the cardio-vascular changes did not precede the renal disease; and the case of James B—, æt. 50 (see Case 18), tends to show that the cardio-vascular changes may be the primary and antecedent disease, and the renal changes subsequent. In this case the kidneys were highly congested and mottled, and a quantity of greyish-looking material was lying amongst this highly congested tissue. These kidneys were large and their cortex was much increased in size, and their surfaces were smooth. The morbid appearances were such as are usually seen when death has occurred in an early stage of acute nephritis, and they appear to show that the kidney disease had probably set in a few weeks only before death. The left ventricle of the heart was much dilated and hypertrophied, and many of the arterioles and capillaries were greatly thickened by the hyaline-fibroid changes. The cardio-vascular disease had, therefore, evidently preceded the acute renal changes.

The arterioles are sometimes much thickened, whilst there is no kidney disease or very little morbid change in the kidneys. In support of this statement we may first mention the case of John C—, æt. 34, who died of aneurism of the aorta. His kidneys were carefully examined, and they presented no signs of disease. The arterioles of the pia mater were much thickened by the formation of fibroid material external to the muscular nuclei. The kidneys were healthy and the arterioles thickened in the case of Sarah S—, æt. 63 (see Case 2); also in the case of Samuel C—, æt. 62 (see Case 19); also in the case of James D—, æt. 77 (see Case 3); also in Case 2.

In other cases there was little morbid change in the kidneys, no morbid change to indicate that their excretory power had been greatly diminished; whilst the heart was much hypertrophied and the vessels much thickened. This is shown in Cases 7, 10, and 20.

Note.—A reply to the above paper was published in the 'Medico-Chirurgical Transactions,' vol. lvi, 1873, entitled "The Pathology of Chronic Bright's Disease with Contracted Kidney, with special reference to the Theory of Arterio-capillary Fibrosis," by George Johnson, M.D.—(Ed.)

DESCRIPTION OF PLATE V.

FIG. I.—The microscopical appearances seen in very granular contracted kidneys. (From a girl aged nine years.)

> The letter A points to six Malpighian bodies which are lying clustered together, and surrounded by a quantity of fibroid material. These bodies contain numbers of fat and other granules. A convoluted tubule indicated by D is seen coming down from one of the Malpighian bodies with its epithelial cells destroyed. The letters B mark tubules which are lined with epithelial cells for the most part healthy. C points to isolated wasted tubules, containing shrivelled, ill-defined epithelial cells, and in some, in place of epithelium, nothing but fat-granules remain.

FIG. 2.—Arteriole much thickened by coarse fibroid changes outside muscular layer; tunica intima thickened also.

FIG. 3.—Tunica adventitia and intima thickened by fibroid changes.

FIG. 4.—Arteriole of the pia mater. Fibroid changes outside the muscular nuclei.

Plate V.

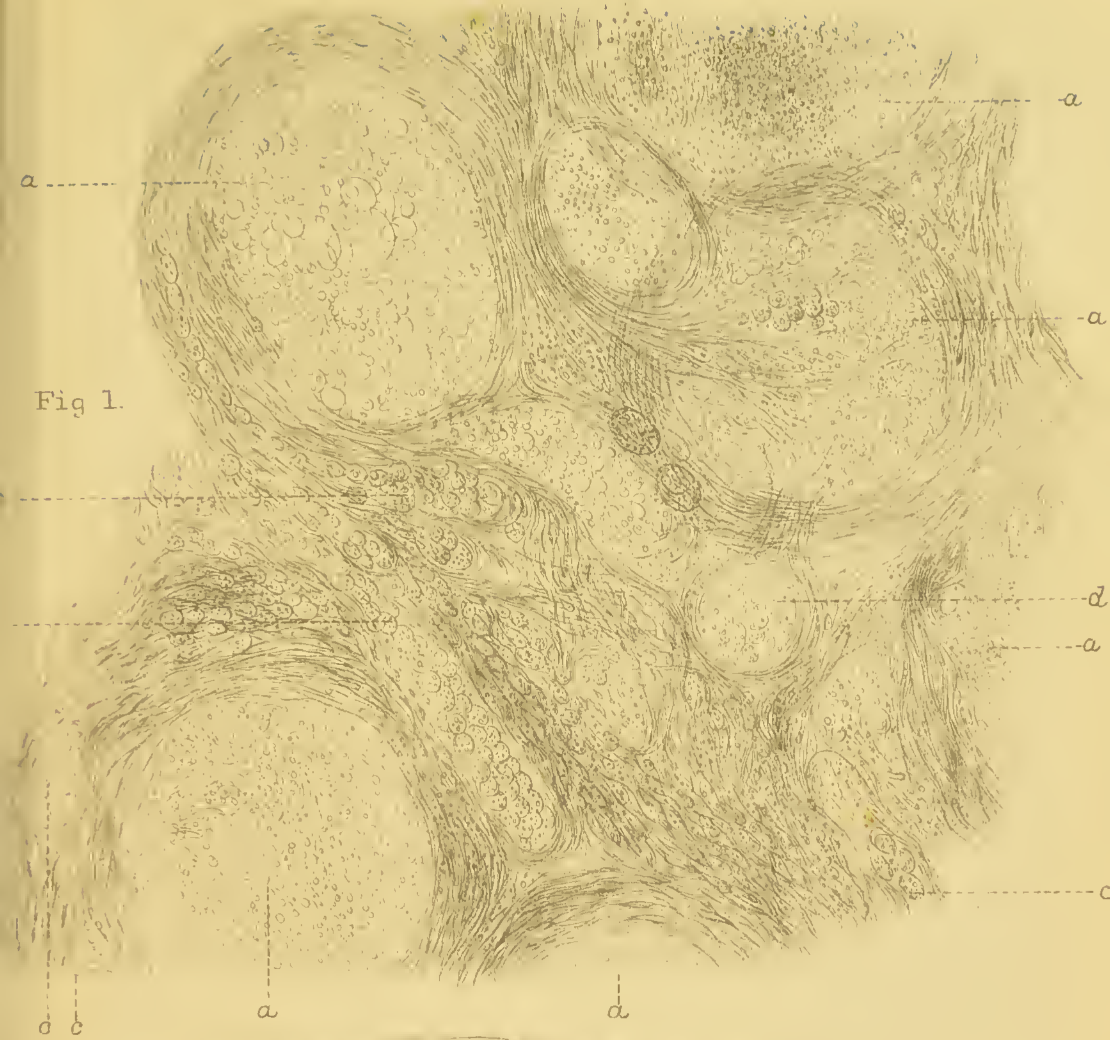


Fig 1.

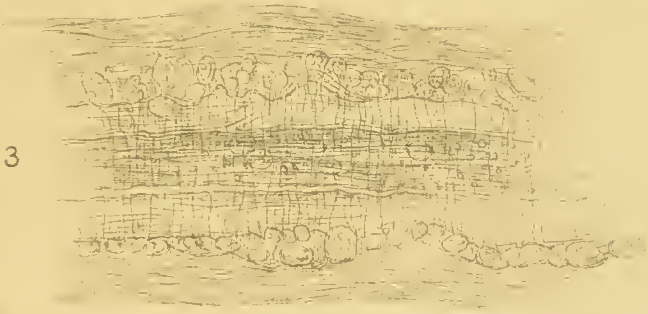


Fig 3

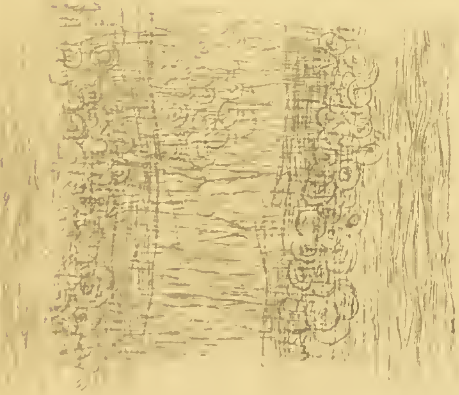


Fig 2.

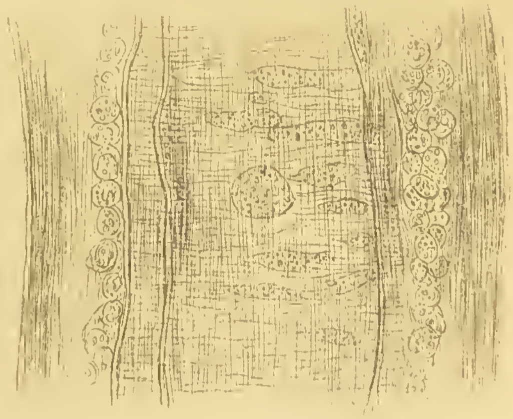


Fig 4.

West, Newman imp.

DESCRIPTION OF PLATE VI.

Fig. 1.—A capillary of the pia mater much thickened by a homogeneous hyaline substance.

Fig. 2.—Capillary very much thickened by a granular substance.

Fig. 3.—Hyaline-fibroid thickening outside the wasted muscular layer.

Fig. 4.—The arterioles in the choroid greatly thickened by the formation of fibroid material outside the so-called muscular nuclei. The dark parts represent choroid pigment.

Fig. 5.—Arteriole of skin thickened by fibroid formation outside muscular layer.

Fig. 6.—Arteriole lying amongst muscular tissue of heart greatly thickened by the fibroid material.

Fig. 7.—Minute artery of the kidney greatly thickened by hyaline-fibroid changes in the outer layer of the vessels.

Plate VI.

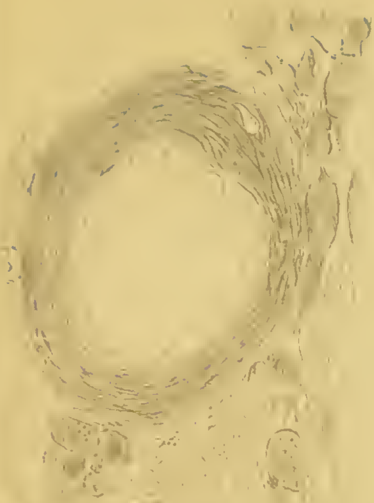


Fig 3.

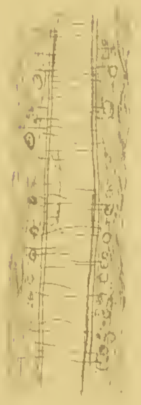
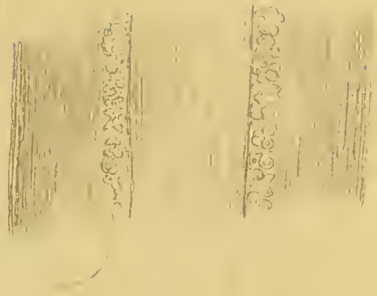


Fig 5



g4.

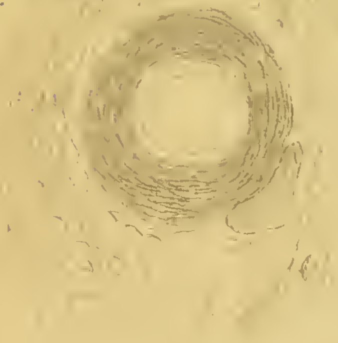
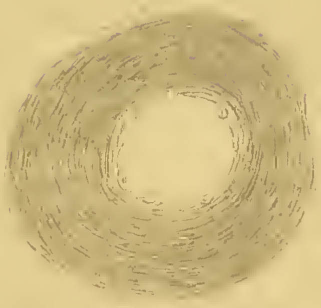


Fig 6.



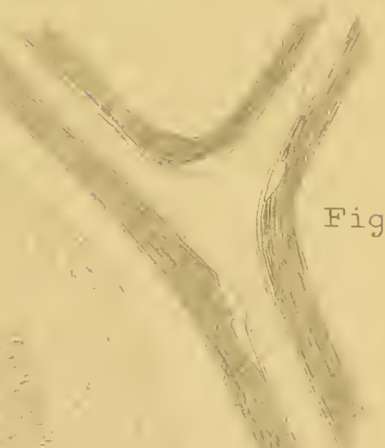


Fig 1

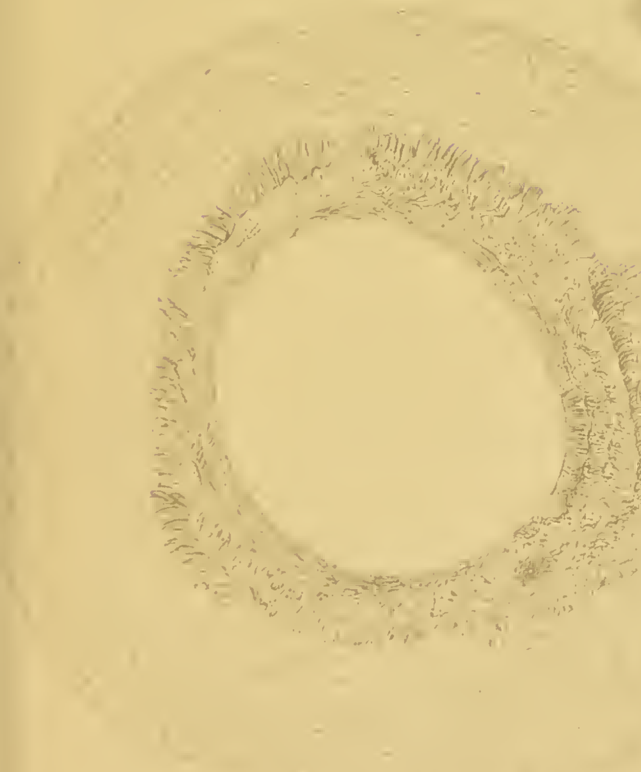


Fig 7.

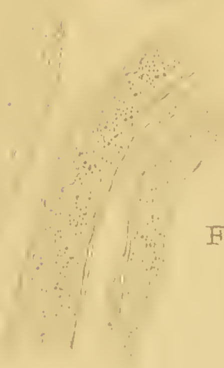


Fig 2.

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CLINICAL LECTURE

ON

CHRONIC BRIGHT'S DISEASE WITH CONTRACTED KIDNEY

(ARTERIO-CAPILLARY FIBROSIS).[1]

THE subject I have chosen for your attention to-day is one for which the clinical wards of Guy's Hospital are particularly famous. To use an expression of our German friends, Guy's Hospital is the "fatherland" of this disease. It was here that Dr. Bright pursued those investigations which have made his name immortal. "Bright's disease" is, however, but a generic expression, and appertains to many affections of the kidney which, though more or less allied by the character of their morbid anatomy, so far as the kidneys themselves are concerned, are probably distinct in their cause and clinical history. It has long been felt that the term "Bright's disease" must either be discontinued, or be more strictly limited to one given pathological state.

The object which I have in view on the present occasion is to make some remarks on that general condition which is perhaps more properly Bright's disease, and which is associated with one form of fibroid contraction of the kidneys. And here it should be observed that the kidney may undergo fibroid contraction and disorganisation, and cause death by uræmia, without being associated with that change in the heart and arteries upon which I desire to discourse in this lecture. In such cases the renal disease is probably due to some hereditary or congenital defect in the kidneys themselves, and is more strictly a local affection; and although the results, so far as the kidneys are concerned, may be very much like, or even apparently identical with, the more morbid conditions arising under other circumstances, what constitutional accompaniments there are, are widely different from such as occur in the disease now spoken of.

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[1] Delivered at Guy's Hospital June 6th, 1872. Reprinted from the 'British Medical Journal,' December 21st, 1872.

The form of chronic Bright's disease with contracted kidney, now under discussion, is, it would seem, a more general affection: it occurs chiefly at or after the middle period of life. Some of its various phases were, no doubt, recognised by the physicians of former times, and vaguely assigned, even so early as the Greek physicians, to special epochs of life, denominated by them "climacterics." It is chiefly between what they would have termed the seventh climacteric and the ninth—that is, between the forty-ninth and sixty-third years—that this morbid condition occurs. I do not quote these terms to give any support to the idea that there are climacteric epochs; but I may affirm that the state of which I am speaking is a mode of decline from the climax of health and vigour of a special kind.

As the facts of disease came to be more and more the objects of inquiry over mere symptoms at the bedside, and as morbid anatomy in support of such clinical observation was pursued more accurately after death, there would naturally be at first a tendency, with increased precision, to a somewhat narrow pathology; for instance, the investigations of the intestines in typhoid fever led Broussais and his followers, as we know, to maintain that fever was a local intestinal affection. A similar error seems to have been committed in respect of this form of chronic Bright's disease. The common change in the urine during life, and the morbid state of the kidneys after death, have so closely confined the attention of physicians, that they have often looked no further than the kidneys and their failing function for the grounds of their pathology, and their pathological views have been limited to the line thus indicated. Now, though no one will deny the almost paramount importance of keeping fully in view the results on different parts of the organism of defective renal excretion, and the tendency to death by uræmia, still we believe it can be no less maintained that the fundamental clinical facts of the disease in question are not included in such limited causation, and cannot be understood by the conditions of a merely renal pathology.

The constitutional form of chronic Bright's disease with contracted kidney, or, as Dr. Sutton and myself have desired to designate it, "arterio-capillary fibrosis," is, it would appear, a primary and general change in the capillaries and arterioles of the body, and corresponds, therefore, in its seat in the adult to the "area vasculosa" in the embryo. And as it is the object of the embryologist to elucidate the changes through which the ovum proceeds upwards to the perfect development of its tissues and organs, so it may be broadly stated to be the object of the pathologist to set forth the steps which lead downwards to death. There are occasions which make it advantageous for the pathologist to enlarge the scope of his considerations by admitting this comparison; for it is obvious that what is generally expressed by the term "vis formativa," whose resultant changes we see in development, is represented by the "vis conservatrix" in the adult, which failing, we have senility and death.

Apart from accidental disturbances in the course of life, we might, perhaps, with due knowledge of the inherited forces, be able to predicate somewhat of the course of their failure. The curve of the ascent and the curve of the decline would, in other words, have some more or less exact correspondence, and we should be able to realise fully the truth that there is not only "a time to be born" and "a time to die," but death might be predicted for a given individual to come in a certain time, say of any particular organ or of any particular system: for instance, we might be able to foretell that the failure would first appear in the nervous, or the vascular, or the secreting system. The form of chronic Bright's disease with contracted kidney is, it seems to us, one example of such (senile ?) decadence.

Its clinical history varies in every case, according to the organ which suffers most and earliest. In one case, sleeplessness, headache, and other slight brain disturbances, may be the first symptoms complained of; in another, the altered condition of the heart may first draw attention; in a third, loss of appetite, dyspepsia, sickness, and failing digestion, are most prominent; in a fourth, diuresis; in a fifth, a liability to bronchitis, with attendant emphysema; in a sixth, a state of general malaise, loss of strength, some emaciation, &c.

These various phases, separately or in combination, constitute the earliest ailments for which the physician is commonly consulted. They are for the most part vaguely referred to a gouty condition; and it must be admitted that the general morbid state of which we are speaking, though distinct from gout, is especially common in those whose ancestors have been gouty. It is often more erroneously referred to suppressed gout. This is less appropriate, since, except the vague flying pains to which such patients are liable, and the occasional deposit of uric acid or urates in the urine, there have often been no phenomena which could properly be termed gouty.

In a large number of cases which come daily before the physician, no doubt the disturbances in question are no more than a mere variation in the general equilibrium of nutrition, which, by due care as to diet and exercise, and the other general conditions of health, are easily corrected; but in a considerable number of persons they are the early signs of a general change in the vascular area, of which disease of the kidneys may or may not be an early and prominent part.

And here I may refer to the evidence supplied by the urine at the outset of chronic Bright's disease with contracted kidney. Hitherto it has been the habit of the clinical physician to take the specific gravity of the urine, and to test it for the presence of albumen. Should the specific gravity be normal (1020 to 1024), and should no change in the fluid be produced by heat and nitric acid, whatever ailments have been present, they have not been referred to the early stages of Bright's disease, but have been explained, as I have said above, by reference to some gouty condition, or to some limited change,—functional or otherwise,—in the part complained of: for instance, if in the stomach, to the food taken; if in the chest, to catarrh from cold. If flying pains have been complained of, they have been referred to neuralgia or rheumatism; if headache, to overwork, &c. But, assuming that we have to do with a case of coming Bright's disease in so early a stage, the following may be considered as not uncommon clinical conditions. The heart may be even already thickened so far as to produce an increased impulse over an increased area. These signs of hypertrophy are somewhat more obvious, as already there may be a loss of subcutaneous fat, and, although not very marked, a further distinct loss of weight.

In a case that lately came before me, the patient, a gentleman aged 53, although he made no mention of emaciation, yet, when questioned, said his weight had declined within a year from 14 st. 11 lbs. to 13 st. 11 lbs. Cardiac hypertrophy under these conditions may have no connection with valvular disease. Indeed, at an early stage both the loss of weight and the hypertrophy of the heart may easily be overlooked; the hypertrophy especially, as the cardiac sounds may have undergone no change, or only a slight loss of distinctness in the first sound.

The examination of the urine at this stage gives varying results. For the most part, there is no discoverable change from health, or only the presence of a faint and doubtful opalescence after boiling and the addition of nitric acid. Even this change may be absent altogether till later on in the disease, or before it becomes constantly present we may find albumen only in the urine passed after food. The presence of albumen may also alternate with the deposit of urates or uric acid. Albumen is often altogether absent when these deposits are present; but the relations of albumen to the deposits of uric acid and urates of soda and ammonia are by no means constant. These deposits and the albumen may occur together in the earlier stages of fibrosis of the kidney. It is by no means uncommon for even careful observers to overlook or lightly estimate the early indications which the urine thus affords of commencing fibroid cachexia.

Although the renal change is probably but part and parcel of a general state of morbid changes, the clinical value of the evidence afforded by the urine in the early stages in many cases is most important. It is desirable to examine the urine passed at different times of the day. The early morning urine may be normal in colour and weight (sp. gr. 1018—1022). That passed at noon may be still without deposits on cooling (1022—1024), with or without a faint opalescence after heat and nitric acid. That passed at 6 p.m. (assuming the dinner to be a late one) may be nearly the same as that at noon. That passed on going to bed may deposit urates, sometimes with, sometimes without faint change from albumen by heat and nitric acid (sp. gr. 1024—1026). It is at this early stage that the symptoms are vaguely referred to disorder of the liver or the stomach.

Those physicians whose minds are made up as to the cause of the cardiac hypertrophy in chronic Bright's disease may insist that even already there may be such a defective excretion from the kidneys as so far to interfere with the physiological relations of the blood to the tissues, that it cannot pass through the systemic capillaries without an increased impulse from the heart, and that the hypertrophy is so caused. But the assumption of such an altered state of the blood as a true cause of the hypertrophy is entirely unsupported by any observations on the blood itself, and is not supported by evidence on the side of the urine itself. The specific gravity, as stated above, may be normal; the quantity and colour may be normal; the cold urine treated with nitric acid may become as usual nearly solid with nitrate of urea; and the only change, and this but occasional, may be a faint opalescence, under heat and nitric acid, due to the presence of albumen. No doubt in a more advanced stage the uric acid and the urates may cease altogether to be deposited as the urine cools. The urine loses colour and specific gravity, and is considerably increased in quantity, though still in general free from all deposits of desquamated epithelium; as the albumen increases, it contains very transparent fibrinous casts, which are complementary of the albumen of the liquor sanguinis, and equally due with it to mere passive transudation. When these changes in the urine have become more marked, and the general cachexia is advanced, the renal affection is set down naturally, if the case is seen at this stage for the first time, as a primary cause of all the attendant changes, cardiac and capillary; but such a view seems to my colleague and myself to be an erroneous one, and to have arisen from a deficiency in our early clinical knowledge of such cases. Nor can this deficiency be easily supplied from hospital practice.

Poor men who are dependent upon bodily labour for their livelihood do not so often present themselves at hospitals in the early stages of disease. It is in private practice that we have to make good our wants in these particulars, and even here the scientific cultivation of medicine suffers by the readiness to explain away the causes of ailments down to the comprehension of the uninformed. It is, besides, to be noticed, that even when our hospitals afford evidence it is indistinct. In an early stage of fibroid cachexia, persons are apt to die of acute disease in the brain or lungs before the amount of renal change is enough to give it any recognised clinical significance. These early deaths take away from us the opportunity of observing the further course in such cases of the renal degeneration which, apart from such accidents, might soon have become a prominent clinical feature, though it escapes attention when less marked; and the subject is dismissed with only this recognition, that the kidneys are coarse. I do not see how we could easily have escaped from the trammels of a narrow pathology, and, if I may be permitted to say so in respect of the matter under discussion, from its vicious influence on practice, if our observations had continued to be limited to the more prominent lesions of advanced "Bright's disease" as they present themselves at the bedside, and even on the post-mortem table.

It is the facts of the early stage of the disease in some cases which amply demonstrate the insignificant part which the morbid state of the kidney, when present at this stage, may then play. I repeat the early stage in some cases, for a further and greater difficulty in the due appreciation of this morbid state arises from the fact that the fibroid changes often come on early, and advance far in the kidneys before other parts are very much affected. Still it is to be remarked that when this is so the renal change is of precisely the same nature, and has the same seat, so far as the several tissues are concerned, as when it occurs early in the capillaries elsewhere. That is, I ought to add, if the disease be the one under discussion; since, as I have before stated, there is a local as well as a constitutional fibroid change of the kidneys. And this brings me to the most important confirmatory fact, showing that mere renal destruction does not produce the state we are discussing. Hitherto it has been assumed and generally admitted that chronic Bright's disease with contracted kidney has always essentially a local origin, and is limited to the kidney in its beginnings, and that the changes in the heart and arterial system are the natural and proper sequelæ of imperfectly depurated blood. Dr. Bright says, speaking of the changes in the heart, "The most ready explanation appears to be that the quality of the blood is altered by the kidney disease. The blood in consequence affects the minute and capillary circulation so as to render greater action necessary to force it through the vascular system." This explanation has been most generally accepted since Bright's time. Dr. George Johnson, to whom we are indebted for much valuable information on renal pathology, fully endorses this opinion, and maintains that there exists a corresponding hypertrophy of the muscular coats of the capillaries compensatory of the cardiac hypertrophy, which acts as a stopcock on the minuter vessels to hinder the afflux of the unwholesome blood to the tissues.

I shall presently have to speak of this change in the smaller arteries (arterioles) and capillaries, and to assert that probably they are not hypertrophied, and that the physiological purpose here mentioned is not founded in fact. But first as to the effect of imperfectly depurated blood—uræmia—on the heart. It seems to have been too readily assumed, in accordance with an experiment by the late Dr. John Reid, which showed that during asphyxia there is an increased pressure on the arterial walls, that a similar state of arterial tension occurs if the blood be imperfectly depurated by the kidneys, and that, this state of tension being continued, hypertrophy of the heart is the result.

But is the effect of blood imperfectly depurated by the kidneys, that which is thus asserted? Dr. Wilks mentions that the heart is free from hypertrophy in many cases of large white kidney. Dr. Dickinson says that simple hypertrophy of the left ventricle is rarely associated with any form of renal disease, except granular degeneration.

Dr. Grainger Stewart found the heart hypertrophied in only about four per cent. of the cases of lardaceous disease of the kidney. To these facts might be added others of a similar kind, such as scrofulous and cystic degeneration of the kidneys. Many of these forms of renal disease are slow in their progress and destructive of the renal function, and they often occur at that period of life when hypertrophy would be readily produced to meet any impediment to the systemic circulation, and yet it does not occur.

A full consideration of all the circumstances makes it, in fact, more than doubtful whether mere defective renal excretion is a sufficient cause of cardiac hypertrophy. It may, perhaps, be objected that the hypertrophy may still depend upon some particular form of renal degeneration. In this, Dr. Sutton and myself are disposed entirely to concur, but not in the sense generally admitted. It is, we believe, when the fibroid change in the kidneys is part and parcel of a fibroid change in the capillaries of the body generally, that the cardiac hypertrophy occurs. Though associated with renal disease, the hypertrophy is not produced by it. To prove how little mere fibroid atrophy of the kidneys, when a local affection, can produce the cardiac and other changes usually attributed to it, I might quote cases of such change in the kidneys in young subjects, in whom, however, after death the heart was not thickened. I might refer to cases also given me by my friend Dr. Moxon; but I prefer on the present occasion to draw attention to one of such cases recorded by Dr. Murchison in the 'Transactions of the Pathological Society,' vol. xxii. And I do this with the more confidence, because in all its important details it corresponds with what I have myself observed to occur from time to time in young subjects of either sex, and is not brought forward by Dr. Murchison in reference to this subject at all.

Walter F—, æt. 18, was admitted into Middlesex Hospital May 9th, and died May 13th, 1871. His father, mother, brother, and sister were alive and well. There had been no more in the family. Ten days before admission to the hospital he was seized rather suddenly with pain at the pit of the stomach, palpitations, and dyspnœa on exertion. Two hours previously he carried a heavy tray of plates fifty yards, and had experienced no inconvenience at the time, though he attributed the attack to this cause. After this he lost appetite, and died on the 13th of the month, four days after admission. The body was well nourished, and showed no signs of chronic disease, and he sank apparently from uræmia. The urine drawn off by a catheter on the day of his admission was limpid and colourless, like water. Its specific gravity was 1007. It contained phosphates, and a decided, though small, trace of albumen. The apex of the heart beat between the fifth and sixth ribs rather tumultuously; the transverse dulness did not exceed an inch and three quarters. On a post-mortem examination the "*heart and blood-vessels were healthy.*" "The only organs diseased," says Dr. Murchison, "were the kidneys, both of which were extremely small, the right weighing one ounce and a quarter, and the left three quarters of an ounce. On section they had a uniform, pale, firm tissue, which on microscopic examination showed very much the same characters as those of an ordinary contracted granular kidney."

I desire to show from this case, as I could from others of a similar kind, that in young subjects renal degeneration from local fibroid changes in the kidney may lead to a fatal issue through defective excretion, without any hypertrophy of the heart being produced. If defective renal excretion alone led to arterial tension as supposed, it is highly probable that, if it occurred in a young subject, in whom the nutritive conditions are active, it would, *à fortiori*, lead to cardiac hypertrophy. It may be objected, that the course of the case was too short for this result; but Dr Murchison says, "Whatever view be taken of the renal lesion, it is quite clear that it had existed a long time prior to death." It would thus appear that fibroid renal disease alone is not a sufficient cause of cardiac hypertrophy. In the case here recorded, the pale and light urine was indicative of extreme renal degeneration. The blood was so imperfectly depurated as to cause a fatal issue. The course of the case must have been chronic; and yet the heart was entirely free from hypertrophy. But whilst, on the one hand, the heart may remain free from hypertrophy, in the long-continued presence of blood so imperfectly depurated by the kidneys as at length to become fatal; so, on the other hand, daily experience shows that, at the period of life when the general fibroid cachexia is most common, cardiac hypertrophy begins and advances to its extreme limits whilst the morbid changes in the kidney are indistinct or early, the kidneys being no more than coarsely granular if obviously affected at all. In illustration, I might direct attention to the frequent occurrence of death by ingravescent apoplexy, at the age of from forty-five to fifty years, where the left ventricle of the heart is often extremely thickened, but the kidneys still unaffected, or, in the early stage of fibroid change, coarsely granular.

The observations which my friend Dr. Sutton has made upon the arterioles and capillaries, afford evidence that in this form of Bright's disease there is a more or less general alteration of the smaller arteries (arterioles) and capillaries throughout the body. To begin with the kidneys themselves, it would appear, in accordance with the views of many other observers, among whom I ought chiefly to name Dr. Dickinson, that the morbid change alluded to begins in the intertubular tissues, and principally in and around the smaller arteries and capillaries. The vascular coats are altered and thickened by the formation of a fibroid or hyaline-fibroid substance. This change affects the tunica intima and the external tunic of the vessels, whilst the muscular coat presents various appearances.

Dr. George Johnson has expressed the opinion that the alteration in the muscular coat is simply one of hypertrophy, and is due to a continued stimulus in the vessels to meet the increased action of the heart, according to the views above quoted. Dr. Beale, who has referred to this matter, thinks with ourselves, that the thickening of the vessels has been too hastily assumed to be one of hypertrophy. Dr. Beale's views on this part of the subject—I mean as regards the vascular changes in the kidneys—are entirely in accordance with what has been seen both by Dr. Sutton and myself. We have found the muscular nuclei often indistinct, and in some cases hardly to be recognised ; and, although there may be an appearance of hypertrophy in some parts of a vessel, it is, as Dr. Beale states, associated with great change and degeneration of the normal tissue in adjacent parts. In confirmation of this, Dr. Beale goes on to say (Dr. Beale 'On Kidney Disease,' pp. 71, 72), "The new tissue added is completely destitute of the properties characteristic of a healthy structure. It is true that we speak of *hypertrophy* of the muscular coat of the intestine and of the bladder, although the tissue may have lost all contractile power ; but it is obvious that the word *hypertrophy* is inapplicable, and ought to be restricted to those cases in which there is not only increased bulk but an increased development of a tissue without impairment of function. In *hypertrophy* of the heart, and of the muscles of the limbs, there is increased formation of healthy muscular tissue, and a corresponding increase of muscular power ; but in the case of these thickened arteries, thickened bladder, intestine, &c., there is an increase of substance depending upon the formation of an abnormal tissue with impaired action or *loss of healthy function* altogether. In the case of the thickened arterial coats there is an increased bulk with altered structure, not simply increased bulk without change in structure (hypertrophy). Careful observation leads me to remark, in the first place, that the muscular fibre-cells are much less distinct than in the normal state. The oval nuclei are to be readily distinguished, and are often increased in size and number, but the contractile tissue has degenerated into mere fibrous tissue, which possesses no contractile power whatever. Secondly, the connective tissue external to the muscular fibre-cells is often enormously thickened, and all indications of the delicate nerve-fibres which ramify in this situation in health are lost. Thirdly, the calibre of the small arteries is considerably reduced, partly perhaps from deposit taking place internal to and amongst the muscular fibre-cells ; partly to the reduced quantity of blood traversing them, but mainly to the increased deposition of new material externally in what was the areolar coat of the vessel."

So far, therefore, as the vascular morbid changes in the kidney go, there is some unanimity amongst observers as to their character, and there is further some agreement as to their being primary and not secondary to morbid changes in the secreting epithelium, as was supposed by Dr. Bright, and is now maintained by Dr. G. Johnson. The early change in the renal epithelium, upon which so much stress has been laid, appears to be largely, if not entirely, due to passive transudation from the vessels, in the act of dying, since similar or even identical appearances are common in the renal epithelium in kidneys otherwise healthy. It is certainly by no means confined to Bright's disease; and it is usual to find it in kidneys which present no signs of lesion except purely mechanical venous congestion.

We believe that the changes in the secreting epithelium will be found to be secondary to the intertubular changes around the vessels, and not, as Dr. G. Johnson believes, primary and essential. This view is confirmed by the condition of the capillaries in other organs. In the lungs, in the stomach, in the skin, in the spleen, in the heart, in the retina, and in the membranes of the brain, the arterioles and capillaries have been found in an abnormal state, from a formation of fibroid or hyaline-fibroid substance similar to that which occurs in the arterioles of the kidney. This morbid change in the vascular system, more or less throughout the body, varies much, however, in different cases, both as to its prevailing extent in any organ, and as to the organ primarily or chiefly affected by it. In some cases, and probably in the great majority, the kidneys are much altered, whilst the heart is at the same time hypertrophied, and the minute arteries and capillaries are proportionately thickened. In others the kidneys may be but little affected, whilst the heart is much hypertrophied, the systemic arteries and capillaries being at the same time thickened by fibroid change.

It is this wide-spread change in the vessels, together with a tendency to atrophy of the surrounding structures, which constitutes, we believe, the essential pathology of "chronic Bright's disease with contracted kidney."

It is a change, as before remarked, which occurs chiefly at or after the middle period of life. And yet the age of a patient is not commonly to be estimated by years. Hereditary predisposition, habits of life, and other circumstances which may lower the vitality of the tissues may induce at a much earlier period than forty-five conditions which do not usually supervene till many years later.

The conclusions at which Dr. Sutton and myself have arrived, are briefly these. 1. That there is a diseased state coming on about the middle period of life, which is characterised by a morbid formation of fibroid or hyaline-fibroid tissue in the arterioles and capillaries. 2. That this is accompanied with atrophy of the adjacent tissues in varying degrees. 3. That this morbid change commonly begins in the kidneys, but may begin primarily in other organs. 4. That the contraction and atrophy of the kidneys is not a cause of the disease, but only part and parcel of the general morbid state. 5. That hypertrophy of the heart is due to changes in the arterioles and capillaries, whereby their elasticity is diminished and so the blood retarded. It is not due to a morbid condition of the blood itself. 6. That the blood may be fatally affected by disease of the kidneys without producing any change in the heart, provided the morbid condition of the vessels alluded to is absent. 7. That the causes which lead to these vascular changes are not yet fully elucidated. 8. That they have an alliance with senile conditions, though probably they are in their nature distinct.

If we turn to the therapeutical bearings of this subject, the discussion of its true pathology does not lessen in importance. At the present time there is a dominant idea in respect of chronic Bright's disease with contracted kidney, that the various functional disorders and lesions which occur in its course are chiefly, if not entirely, connected with a *materies morbi* in the blood, to which such disorders and lesions are chiefly referable. The therapeutics of the day are largely directed according to this idea. Like most theories respecting the *materies morbi* of different diseases, whether we regard the elimination of the poison of cholera, the neutralisation of an acid in the blood in rheumatism, the destruction of it by colchicum in gout, or the antidote to some septic poison in fever, the theory that all or most of the symptoms in chronic Bright's disease are due to uræmia is frequently fraught with dangerous and destructive effects in practice.

I call vividly to mind the unhappy results of treatment so directed : the vapour-baths and antimonials of former times for eliminating through the skin; the compound jalap powder and elaterium, to call forth the vicarious functions of the intestinal mucous membrane, *et cætera similia,*—such or similar treatment tended, no doubt, to exhaust the already failing nutritive powers.

Although, in respect of the uræmic state, the faulty condition of the blood and its treatment by eliminants cannot be neglected, still it must be admitted that there is an important distinction between the uræmic state and its consequents, and that general fibroid cachexia (?) which exhibits itself in the arterioles and capillaries of the different organs, as now under discussion. The two may no doubt be combined, and indeed are often combined, in the same individual; but still, for therapeutic purposes, it is of no small importance to the sick man, that as exact a perception as possible of the distinction here insisted on should be acquired.

Whilst we cannot leave out of consideration the changes in the blood, neither can we safely forget that there are important antecedent and comcomitant tissue changes which are independnt of mere uræmia.

I have not time fully to discuss, even if it were my duty to do so on this occasion, all the clinical bearings of this subject. My colleagues, your present teachers, are as fully alive to the necessity of extending our views on this subject as I myself can be. They will, I am sure, tell you that many of the diseases of the brain, of the lungs, of the heart, and of the stomach, are associated with, and form part and parcel of, this great malady. When treating of apoplexy, they will say that over and above the cerebral affections which are due to uræmia, and which were first of all so well described in this place by my late colleague, Dr. Addison, the brain tissue is liable to subacute or chronic lesion, from disease of its capillary vessels; that acute pneumonia, or bronchitis, at the period of life of which I have spoken, is often due to already commencing vascular disease; that dyspepsia, and especially the so-called gouty dyspepsia, is not the result of a *materies morbi* floating in the blood, but of a subacute or chronic degeneration of the mucous tissue.

Here, as elsewhere in the therapeutics of disease, the limitation of our thoughts to the conditions of the moment, or to the special organ affected, cannot but be productive of either fruitless or dangerous practice. It is always dangerous to rest in a narrow pathology; and I believe that to be a narrow pathology which is satisfied with what you now see before me on this table. In this glass you see a much hypertrophied heart, and a very contracted kidney. This specimen is classical. It was, I believe, put up under Dr. Bright's own direction, and with a view of showing that the wasting of the kidney is the cause of the thickening of the heart. I cannot but look upon it with veneration, but not with conviction. I think, with all deference to so great an authority, that the systemic capillaries, and, had it been possible, the entire man, should have been included in this vase, together with the heart and the kidneys; and then we should have had, I believe, a truer view of the causation of the cardiac hypertrophy, and of the disease of the kidney.

ON CHANGES

IN THE

SPINAL CORD AND ITS VESSELS IN ARTERIO-CAPILLARY FIBROSIS.[1]

Before entering upon the morbid changes which are the subject of this communication, it may be permitted us to state some general conclusions on arterio-capillary fibrosis to which our observations have led us.

In May, 1872, we recorded in the 'Transactions' of another Society a series of observations on the morbid state commonly called chronic Bright's disease with contracted kidney, and affirmed that there are (1) not only the well-recognised, and we may say, notorious cases in which the kidneys are contracted, the heart much hypertrophied, and the vessels diseased, but there are (2) others in which the kidneys are but slightly affected, and yet in which the heart is equally hypertrophied and the vessels diseased, and (3) other cases in which the heart is hypertrophied, the vessels diseased, but without disease of the kidney of the kind in question, or merely the congestion of the dying. In all these three classes of cases we have observed fibroid changes in the arterioles, capillaries, and interstitial tissue of various organs. On these grounds we expressed the opinion that the pathology of the state commonly called chronic Bright's disease with contracted kidney was not essentially renal, and that for its full comprehension a wider investigation of concomitant or even antecedent changes in other organs was called for. Since the time named we have prosecuted these investigations more or less continuously throughout the several organs—stomach, spleen, liver, lungs, heart, cord, brain, skin.

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[1] By Sir William Gull, Bart., M.D., and H. G. Sutton. Reprinted from the 'Pathological Society's Transactions,' vol. xxviii, 1877, p. 361.

If further inquiry should establish, as it seems to us assured that it will, that after the middle period of life, there is very commonly a pathological condition of the body which leads to fibroid changes, not only in the kidneys, but more or less generally in other organs, then we may conclude that the renal affection, being of the same kind and character, is probably but a more pronounced local expression of a general disease or degeneration.

Clinical medicine from this point of view would recognise the significance and bearing of many now supposed unimportant ailments; and might find that these ailments are signs of commencing tissue-changes of the kind in question, springing up in one or more of the several organs, it might be in advance of renal changes, and foreboding their advent. But lest we should be misunderstood as too much limiting this inquiry, we would state that our investigations lead us to think that these tissue-changes may in some cases result from the renal disease; whilst in others they may follow the renal changes in respect of time, but not be dependent upon them, but upon a general cachexia of which the renal disease is part.

Everyone will admit that the progress of pathology must be made by retracing the steps which lead to morbid anatomical results. In chronic Bright's disease with contracted kidney, the kidneys and the thickened heart, the two most prominent features, have mostly occupied and satisfied the attention, whilst antecedent or attendant changes in the other organs have been but little considered; or, further, indeed, when such changes with hypertrophy of the heart have occurred without the prominent lesion in the kidneys, they have been too much regarded as isolated facts. We believe it will be proved that these collectively indicate in common a state having arterio-capillary fibrosis as its basis.

Respecting the objection which was raised by Dr. Johnson, that what we had regarded as a pathological change in the arterioles and capillaries was a merely artificial result, we have only here to state, with due respect to him, that continued investigations have but strengthened our views; and the observations we have now to submit on the cord will probably leave little doubt in the minds of others that the changes in the arterioles and capillaries are morbid. As to the hypertrophy of arterioles, we may say, as we did in our earlier communication, that though the muscle in some of the larger arterioles especially seemed to be increased, yet we are still sensible of the difficulty of giving a true interpretation to such an appearance. As to the question whether the muscular layer of the arterioles in any particular instance be hypertrophied, assuming the several elements to be normal, or whether the thickened appearance in such a case is the result of unusual contraction of the vessel, we have not, as we say, been able fully to satisfy ourselves. The problem is beset with special difficulties, and obviously greater ones than can arise in determining whether the arterioles be the seat of morbid changes in their tissues or not. Moreover, we know of no observations showing that the muscular layer has a constant thickness in arterioles of equal calibre.

Nor is it always easy to say whether the adventitia of an arteriole is thickened if it be separated from its surroundings. At all events, in a doubtful case, we are much aided by seeing how the apparently thickened adventitia is in continuity with the increased connective tissue about it, and how the fibroid changes spread from the vessels to the surrounding textures.

But whatever conclusion shall be arrived at respecting the hypertrophy, we maintain that the muscle layer in many arterioles in chronic Bright's disease and the general state associated with it, is atrophied, and associated with a hyaline-fibroid change in arterioles and capillaries.

In submitting our observations on the spinal cord, we desire to add that we have extended our inquiries to other organs, and are prepared to show that there are similar tissue changes in various seats and amount throughout the body in this morbid state.

This communication we regard as but part of a series growing out of our former inquiry, and enlarging our conclusions recorded in 1872.

We believe that many, if not most, of the textural changes in the cord, will be seen to be similar in kind to those which characterise the fibroid contracted kidney. Hereafter we purpose to show from observations already made that the same character of lesion occurs in stomach, spleen, heart, lungs, brain, skin, &c.

The sections of the spinal cord which we now bring forward, are prepared in the usual way, hardened by chromates, stained with logwood or carmine, or anilin black, and mounted in Canada balsam.

Before entering upon the morbid changes in the cord in cases of arterio-capillary fibrosis, it may be useful to recall some particulars of the normal histology.

The surface of the cord is bounded by connective tissue, which is simply part of its pia mater, and processes of the same penetrate at many points into the white matter. Many of the larger processes (septula) pass in a straight direction inwards; but, in doing this, they give off branches, and enclose groups of nerve-tubules, making it not difficult to imagine that the columns of the cord are comparable to a fasciculus of medullated nerves, bound together by connective-tissue sheaths which are the remains, or at all events the representatives, of the neurilemma. The thinner branches of the septula give off still more slender processes, and the finest of these pass between and separate individual tubules.

By this branching and communication, a connective-tissue plexus is formed, in the meshes of which the nerve-tubules lie as in a stroma. Gerlach estimates that the most attenuated divisions do not exceed ·008 of a millimetre in thickness. The septula of connective tissue in the lateral columns near the grey matter are thicker than in many other parts, and thicker in the posterior than in the anterior columns, and especially near nerve-roots.

Opinions have differed as to the structure of the septula, but Gerlach says the larger are made up of slightly sinuous fasciculi of the very finest connective-tissue fibrillæ, which run mostly horizontally to the long axis of the body. Our observations support his opinion (see Pl. VII, figs. 1 and 2.)

If the connective tissue from the septula be traced inwards until the finest divisions of the plexus are reached, we see, in transverse sections of the cord, nuclei lying here and there between the nerve-tubules. Immediately surrounding these nuclei is a small amount of protoplasm; and from this protoplasm radiate two, three, or more, exceedingly slender caudate processes, which divide and subdivide and pass between the tubules. It is these caudate fibrils which constitute the finest intertubular branches of the connective tissue (see Pl. VII, fig. 2). These connective-tissue nuclei with their caudate fibrils are especially well seen in the columns of the cord, near to the grey matter (see Pl. VIII, fig. 1), and with their protoplasm are particularly distinct in the anterior columns, near to the anterior median fissure. But they may be found in almost every part of the white matter. They are better brought out by logwood dye than by carmine. In addition to these elements there is a finely granular and homogeneous substance (neuroglia) which embeds the tubules. This neuroglia is considered by some observers to be simply a modification of the connective tissue, interlaced by elastic fibres.

A line bounds the outer edge of the medullary sheath, and separates it from the neuroglia. We are disposed to agree with those who look upon the concentric appearance commonly seen in the medullary substance of the nerve-tubules, especially in chromic acid preparations, as an artificial production.

Of the grey matter, for our purpose, we need not say more than that the nerves are surrounded by neuroglia, but without the fibrillar connective tissue as in the white matter; and that a connective-tissue plexus supports the columnar epithelium of the central canal. Outside this is a very fine nerve-plexus, and a number of scattered spherical bodies, which are thought to be connective-tissue cells. The nature of some of these bodies is uncertain. The distribution of the vessels in the cord is not yet fully determined. Artificial injections show arterioles and capillaries ramifying in the septula of connective tissue. These vessels are often seen in the septula naturally injected. The capillaries and arterioles of the grey matter are generally seen without difficulty. The arteriole and vein on each side of the central canal are familiar objects.

We have given these brief particulars of the healthy cord, with the object of rendering our description of the pathological alterations more intelligible.

These alterations will perhaps be better understood if we describe first the slighter and more recent, and subsequently the more advanced and complicated changes.

We first describe (Case 1) the changes which we think are due to œdema—simple exudation as part of a more or less general œdema. In a section of a dorsal cord[1] are seen many small homogeneous masses, well stained; these vary much in size: some are only as large as the area of one nerve-tubule, while others occupy the area of two or three. The edges of these masses are not always well defined; they fade off gradually into the adjoining structures. As many as twenty to thirty of these masses can be counted at one time in the field (150 diameters). They are present in this case in all parts of the white matter, but are more numerous in the deeper parts of the columns. These masses are simply diffused amongst the nerve-elements and connective tissue. This is evident by noticing that although the homogeneous stained substance may conceal the axis-cylinders and medullary sheaths, yet, if the focus be altered, these structures are seen lying, seemingly little or not at all changed, amongst the stained homogeneous matter. Similar homogeneous substance is noticed also collected around some of the capillaries, and so much so that the wall of the vessel is buried in it. Still it is to be noted that most of the collections are not in immediate contact with arterioles. Besides these homogeneous masses, there is also a hazy substance, probably of the same nature as the above, but less in amount, and therefore more faintly stained, apparently pervading many of the medullary sheaths, and even extending into axis-cylinders, and also a few collections of yellow

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[1] Case of W. L— (Pl. IX, figs. 1 and 2).

(hæmatin) granules. These defined homogeneous masses correspond to what have been called colloid bodies (see Pl. IX, fig. 2, β). The fibrils of the connective-tissue cells appear thickened in many parts. Whether this apparent thickening be due to exudation around the fibrils, or to an increase of the substance of the fibrils themselves, may be open to question. The protoplasm around the connective-tissue nuclei appears increased, but the nuclei themselves of the connective tissue appear in this case mostly normal, but some seem swollen and are aggregated together in parts in twos and threes. The vessels are for the most part not noticeably thickened. Some axis-cylinders in one of the sections of the anterior column are enormously enlarged by exudation, or, at least, by some material which took the dye well (Pl. IX, fig. 1).

In the spinal cord we next refer to (Case 2, W—, æt. 42: autopsy showed kidneys granular and contracted; left ventricle of heart hypertrophied; old hæmorrhagic changes in brain, &c.), there are changes (simple exudation) similar to the above, but the vessels and connective tissue are thickened by fibroid material. In the anterior columns especially, some of the fibrils of the connective-tissue plexus are thickened by fibroid material, and some of the adjoining nerve-tubules are completely concealed, as if they had been destroyed by it. There are many small centres of this fibroid thickening. Portions of the columns appear healthy, but the greater part is evidently the seat of morbid change. Most of the connective tissue is free from the fibroid thickening, but granular matter is exuded along its fibrils, or the fibrils themselves are swollen by exudation. There are collections of hæmatin granules in one of the posterior columns, also showing exudation. Together with these changes there are numerous masses of homogeneous (stained) material, mostly spherical in shape. Their outline is not well defined. Their substance seems diffused amongst the nerve-tubules and connective tissue. In portions of the column these masses are very numerous. Here and there they are more circumscribed, and they have the appearance of being bounded by a very thin wall, suggesting that the material is accumulated within a nerve-sheath, and distends it. Owing to the accumulation of this exudation, the connective-tissue fibrils, instead of forming thin partitions between the nerve-tubules, as in healthy cords, appear spread out, rendering the nerve-tubules very indistinct. They appear to be embedded in a granular homogeneous substance. Some of this substance could be directly traced as effused from the vessels, for in one part of a section a vessel is seen cut across, and surrounded by hyaline homogeneous (dyed) material. Some of the arterioles of the dorsal portion are very noticeably thickened by fibroid material, and it was particularly observed that whilst the tunica intima of one of them is normal, and some of its transverse muscle-cells normal, other muscle-cells are indistinct, as if much shrunken; and outside the muscle layer there is a homogeneous substance bounded by coarse and dense-looking fibres, amongst which are spindle-shaped nuclei. In some of the larger arterioles the muscle-cells appear larger than normal (hypertrophied ?), but in other parts of the same vessel they are apparently atrophied and reduced to mere granules. The walls of some of the capillaries look simply coarse, but others are evidently much thickened, for the walls of some are thicker than the diameter of their lumen.

The above details denote chronic changes in the arterioles, capillaries, and connective tissue, with recent exudation.

We may now refer to one more cord (Case 3, of Ann C—, æt. 52: autopsy showed granular contracted kidneys; dilated hypertrophied left ventricle; atrophied skin and spleen; arterial disease), in which there are morbid changes resembling those above. The adventitia of the arterioles is thickened and coarse, with here and there an excess of elongated nuclei in it. The walls of some of the arterioles have a hyaline appearance also. Much of the connective tissue seems abnormally rigid and coarse, and, as in the two other cords referred to, there are appearances indicating that there has been recent exudation between and into the nerve-tubules. Therefore a great many of the septula throughout the cord, even where they are not markedly thickened, have an abnormally granular aspect; and in parts, homogeneous material clouds the medullary sheaths and the axis-cylinders, whilst there are numerous spherical homogeneous stained (colloid) masses, such as were seen in the two previous cases.

The changes in the connective tissue of the above-named cords are slight compared with those observed in the cords to be now described, and some of the changes are so minute that we cannot appreciate them by the lower powers of the microscope, though by comparison with a healthy cord and under higher powers the changes are very obvious. The fibroid thickening in the adventitia of some of the vessels (W— and C—) indicate chronic change, though the very large quantity of homogeneous and very finely granular material scattered largely through these cords, is regarded as the product of recent serous exudation. We were led to this opinion from the appearance and arrangement of the material itself in these cords, and from other facts which have come under notice.

We have already stated that a very large portion of this homogeneous and granular material is diffused through the cord-structures, without any abrupt margin; but some of it is sharply bounded, not apparently by any new structure, but by being moulded to the outline of a tubule; but it is conceivable that the boundary around some of it may have been the wall of a lymphatic vessel, presuming such vessels exist in the cord. These so-called colloid bodies are probably, as we have said, but simple exudations of albuminoid material. Similar hazy, homogeneous material, we have found, more or less hyaline, but in very much smaller quantity, in parts of a healthy cord; for instance, in that of a boy suddenly killed. If it be albuminoid matter the product of simple serous exudation, it might be expected that it would be found in minor degree, even in healthy cords, especially where death occurs rapidly by injury; for if the body be well nourished, then, especially during very rapid dying, the venules and capillaries of the cord, as of other organs, must be abruptly and unduly distended as the pulmonary circulation is suddenly arrested; consequently serum escapes more or less from the distended vessels into the substance of the cord, liver, kidney, and other organs. Microscopical examinations of the organs of persons accidentally killed in health, teach that at one time serum alone escapes, at another serum together with leucocytes, with or without red corpuscles. But neither in healthy cords, nor even in all cords from cases of Bright's disease, have we seen such large and well-defined collections of granular and homogeneous material as observed in the three cords above described. Again, to show that these homogeneous collections hitherto called colloid masses are sero-albuminoid exudations, we may state we have observed, where the brain substance has been contused by accident, as in fractured skull, that similar masses are scattered in very large quantity, and similarly diffused into and amongst the nerve-elements. For instance, sections of the pons and medulla oblongata taken from the body of a healthy young man, killed by fracture of the base of the skull, more especially of the pons, were thickly studded with an enormous quantity of these homogeneous masses, and here and there hæmatin granules. Again, where the cord has been injured by fracture of spine, we have found these homogeneous masses in very great quantity in the cord substance. Further, in cases of acute paraplegia (myelitis) a large quantity of this homogeneous material is found lying around the vessels, and disseminated from them into the surrounding structures, with increased nuclei and leucocytes.

From these facts, together with the appearance and arrangement of these colloid masses, we are led to regard them as merely albuminoid exudations, the water of which has been removed in course of preparing the sections; and the very ready way in which the material takes dye, tends to show, as we have already once remarked, that it was at the time of death plastic. It is, moreover, to be noticed that there is not with such exudation a great increase of the nuclear bodies, or corpuscles, to indicate that the exudation is inflammatory. Still it is in much greater quantity than is found in the mere passive congestion of healthy cords. We are, therefore, led to conclude that this condition is simple œdema of the cord, coincidentally, it may be, with œdema of other parts of the body. Naked-eye examinations show that the membranes of the cord, especially of the lumbar region, are not infrequently œdematous in cases of intertubular nephritis. In giving this account of these bodies we ought to state that somewhat similar colloid masses may, as is supposed, be produced by dissolution of nerve-tubules.

We may now pass on to describe morbid changes more marked and more advanced.

First there is thickening of connective tissue by broad stream-like collections of hyaline homogeneous substance, this being in such amount as to compress and invade the nerve-tubules, so that the axis-cylinders are destroyed or invisible. Such a condition is more than mere œdema, but still short of actual myelitis. Secondly, there follow exudation and multiplied connective-tissue nuclei and leucocytes—myelitis.

In Case 4,[1] in the dorsal portion of the cord the connective tissue is seen much thickened by a homogeneous substance, studded with an excess of nuclei, clustered in parts into twos and threes; the walls of some of the arterioles and capillaries are much swelled and markedly hyaline; their outline is partially concealed by this hyaline (dyed) homogeneous substance, which infiltrates the surrounding connective tissue, and spreads away from the arterial wall amongst the surrounding nerve-structures. (See Pls. X and XI.)

This homogeneous (dyed) substance extends into some of the medullary sheaths, but the axis-cylinders appear to a great extent unaffected, though some seem invaded by it. It is also very noticeable that there is increased nuclear formation in the grey matter. Together with these recent changes, there are also indications of chronic thickening by a fibroid substance of the adventitia of the arterioles. The muscle-cells in some arterioles are inappreciable.

In the columns of cord of another case (Case 5[2]) there is similar evidence of myelitis and indications of chronic change. The connective tissue of the columns of the lumbar portion is thickened throughout with an excess of nuclei. This thickening is largely produced by an exudation of granular and hyaline homogeneous (dyed) material in and around the the connective-tissue nuclei and fibrils. The connective-tissue nuclei are increased or leucocytes aggregated. Also spherical bodies (colloid masses) are scattered here and there. Some vessels (arterioles) are much thickened, their walls even thicker than their lumen, and nearly all have the characteristic hyaline appearance.

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[1] Case 4, of James B—, æt. 47. Autopsy showed granular contracted kidney, with indication of recent acute nephritis; emphysema; dilated and hypertrophied left ventricle; great contraction and granular condition of mucous membrane of stomach; atrophied brain. Pericarditis and pneumonia.

[2] Case 5, of William Geo. H—, æt. 69. Autopsy showed simple fracture of femur; granular contracted kidney; hypertrophied heart, weighed 18 oz. Lungs emphysematous; pleuro-pneumonia at base of one. Atrophied

The outline of some of the arterioles is coarse, with spindle-shaped nuclei scattered along it, but these are seemingly not in any decided excess. The connective tissue extending between nerve-tubules from the adventitia of some of the arterioles is markedly thickened by felt-like and homogeneous substances. These appearances seem to denote that the vessel walls and the connective tissue of the cord are thickened by some chronic fibroid change as well as swelled by some recent exudation.

Another very striking feature is the puckered condition of the surface of this cord. At the depressed parts tracts of thickened connective tissue extend from the surface into the substance of the columns. In reference to this condition we may remark that though in healthy cords there is usually slight depression, where the vessels pass in at the surface, yet the contrast between the abnormal puckering above described from thickened connective tissue, and that which normally occurs, is striking. In these thickened fibroid tracts are arterioles, in the thickened walls of which is an excess of nuclear bodies. The connective tissue about the posterior roots especially is much thickened, and also contains a great number of nuclear bodies and homogeneous colloid masses; and the surface of the cord corresponding to this part is still more abnormally puckered and uneven. This fact of irregular puckering and contraction, associated with thickened connective tissue, seemed to us so important that,

spleen, with thickened connective tissue. Atrophied brain. No œdema. No indication in kidney of acute nephritis.

before determining that it had a pathological value, a careful comparison was made with healthy cords.

The changes in the above-named cords are distinct, however much difference of opinion there might be as to their significance, but they seem small in degree when compared with the changes we now come to.

In the dorsal portion of the cord of Case 6,[1] but most of all in the posterior columns, the connective tissue almost throughout the white matter is enormously altered. The septula and their finer processes are replaced by a dense homogeneous substance. This new material, which has taken dye well, is studded with many large (swelled ?) nuclei. These are in great excess, and aggregated into small groups (see Pl. XII). In the posterior columns this dense substance is in greatest quantity. Many tubules are embedded in it, and their axis-cylinders only seen. Others would seem to have been entirely replaced by the exudation. But even where this material occurs in greatest quantities, some medullary sheaths and axis-cylinders, even in its vicinity or surrounded by it, remain almost normal. The arterioles and capillaries are seen very greatly thickened by homogeneous or faintly fibroid material. Capillaries and very fine arterioles with much thickened walls are seen surrounded by great quantities of this same material, which radiates into the surrounding connective tissue, compressing and invading the nerve-tubules (see Pl. XVIII).

We now come to what seem to us more chronic changes. In the optic thalamus in Case 7[2] there is fibroid thickening, forming a kind of scar, embedding hæmatin crystals, evidently the remains of old hæmorrhage. The septula of the cord in this case are much thickened by a fine felt-like substance. The changes here seen recall to our minds that the French pathologists have described similar fibroid thickening in tracts of the cord in cases of old cerebral hæmorrhage, and called it "descending sclerosis." The morbid growth in this cord had destroyed many of the medullary sheaths and axis-cylinders.

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[1] Case 6, of Henry G—, æt. 36. Autopsy showed dilated hypertrophied left ventricle; atrophied brain; syphilitic changes in liver (?); kidneys simply venously congested.

[2] Case 7, of T—. Autopsy showed old hæmorrhagic changes in the form of ochrey matter in the corpus striatum, recent blood-clot in the one hemisphere; dilated hypertrophied left ventricle; granular kidneys.

In another case (Timothy K—[1]) the chronic fibroid change, though very marked, was confined to numerous small areas of the columns (*sclérose en plaques*). In the dorsal portion of the cord, the part principally examined, there is much of this morbid change, but it is greatest near the surface of the cord in the vicinity of the posterior roots. Even with half-inch objective the connective tissue seems to be here and there much thickened, though there is a good deal of healthy nerve-structure remaining. It is one of the most noticeable features of this cord that the connective-tissue nuclei are surrounded by a quantity of fibroid material, as if they were centres of this thickening. Such is the interpretation we adopt. Examining this new material further (× 250), the nuclei are seen well stained and apparently swelled, and extending from them is an exceedingly delicate felt-like substance, spreading out and invading and destroying adjoining nerve-tubules; though in the vicinity of these destructive processes there are normal axis-cylinders and medullary sheaths (see Pl. XIII); the connective-tissue plexus between them has a coarse appearance. In some parts both connective tissue and nerve-structures seem normal. This is especially so in the lateral columns. This new material is not homogeneous, as we have already said, but seems to be made up of extremely delicate fibrillæ, which can be best expressed as felt-like. Many axis-cylinders appear abnormally large (hypertrophied ?). Some of the arterioles and capillaries are surrounded by a large quantity of new (felt-like) material; some are seen to be so much thickened that the thickness of their walls is double that of the lumen. The hyaline appearance is well marked. There are a number of homogeneous (colloid) stained bodies scattered over the section. These are very noticeable objects in the centre of the thickened masses.

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[1] Case 8, of Timothy K—, æt. 43. Died November 22nd, 1875. Autopsy showed brain convolutions much atrophied; vessels atheromatous. Heart weighed 20 oz.; left ventricle dilated and hypertrophied; valves normal; lymph on pericardium. Lungs collapsed at bases. Kidneys small and granular, weighed 3½ oz. each. Liver and spleen healthy. Muscular atrophy well marked in muscle of thumbs and interossei and muscle of forearms.

Tracing further these chronic changes, we may describe next the alterations seen in Case 9.[1] In the anterior columns, and especially near the posterior roots, but in other parts also, the connective tissue is much thickened. Many small foci of thickening are seen as in the preceding case, and in the centres of these are nuclei, with much fibroid material around them, and in many parts where this has accumulated the nerve-tubules cannot be distinguished, or only the axis-cylinders, which are so shrunken as to be scarcely recognisable. With these changes there is still a great deal of healthy nerve-substance. Arterioles and capillaries in this cord are also much thickened by fibroid material.

We now adduce evidence of more advanced and more extensive fibroid changes in cord—diffused sclerosis.

We take a case (Case 10) in which there was progressive muscular atrophy and distinctive cardio-renal changes.[2] In the dorsal portion of the cord there is extensive morbid change, more especially in the posterior and lateral columns. The larger septula near the periphery are much widened, and it seems evident that the thickening has extended from the surface inwards. Some of these septula are three or four times thicker than normal; this increased thickness is produced by fine (felt-like) fibroid substance, without large nuclei. From each side of these widened septula thickened branches of fibroid material are given off; and in some places near the surface these are so broad that they seem to have coalesced and formed an uniform mass of fibroid substance, in which scarcely a nerve-tubule can be seen (see Pl. XV). In other places the thickened branches have not coalesced, but the growth has extended thickly round the nerve-tubules. Here and there is a normal nerve-tubule embedded in this fine felt-like thickening; or the medullary sheath may be gone, leaving only the axis-cylinders; or there is no decided trace of nerve-tissue remaining, being replaced by this new fibroid material. At a little distance from the surface of the cord, these very broad septula have their fibrillar ramifications replaced by broad lines of homogeneous or fine felt-like substance, surrounding each nerve-tubule. Nerve-tubules are seen embedded in ring-like masses of this substance (see Pl. XIV). It is instructive to notice that in some parts there are numbers of connective-tissue nuclei scattered as in healthy cords. They are well stained, but there is a much larger amount of protoplasm than normal around them. It is further noticeable that not only has the protoplasm increased around the nuclei, but these latter have multiplied. The condition of the grey matter we consider undetermined.

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[1] Case 9, of James C—, æt. 56. Autopsy, February 5th, 1874. Dilated and hypertrophied left ventricle, heart weighed 22 oz.; no valvular disease; suppurative nephritis; brain very slightly atrophied.

[2] Case 10, of William S—, æt. 40. Autopsy, March 17th, 1873, showing muscular atrophy; granular contracted kidney; indication of acute nephritis; dilated hypertrophy of left ventricle; no valvular disease nor adherent pericardium; brain slightly atrophied.

In the most advanced stages of the changes we have thus called attention to (Case S—), broad masses of uniform fibroid substance are to be seen completely replacing areas of nerve-tubules, and sending off thick processes of the same fibroid material which surround and embed some nerve-tubules, and have destroyed others. This is well represented in Pl. XV.

Whilst extensive atrophy of the nerve-tubules thus occurs with this deposit in some parts, in others the axis-cylinders are enormously swollen or hypertrophied.

The vascular sheaths are also seen surrounded by a large amount of the same fibroid substance.

We adduce two more cases (P— and M—) where the chronic fibroid change was very much advanced. Both terminated in paraplegia. Sections of the lumbar portion of the first (P—,[1] Case 11) show the connective tissue in many parts of the white matter very much thickened by a felt-like fibroid material. This is especially marked in the posterior and lateral columns, but it is present also in the anterior columns. The thickening is very irregularly distributed, leaving some portions of the columns comparatively healthy, certainly much less affected. Many of the septula are extremely thickened by this fibroid substance, which extends from them along the course of the connective-tissue plexus, invading some nerve-tubules, rendering their axis-cylinders very indistinct (shrunken) or inappreciable. Many of these thickened masses send off branches which intercommunicate. In their meshes are medullary sheaths, seemingly swelled up and disorganised, and the axis-cylinders scarcely or not at all recognisable (see Pl. XVI). There is also a cloudy homogeneous material scattered here and there, seemingly exudation material, faintly stained; also spherical homogeneous masses, so-called colloid bodies. Further, in portions of the fibroid material there are a number of spherical nuclei, which appear to be newly-formed connective-tissue corpuscles.

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[1] Case 11, of George P—, æt. 58. Autopsy showed atrophy of brain; vesicular emphysema, with broncho-pneumonia; kidneys faintly granular; cystitis; spleen small. Heart weighing 11½ oz.; left ventricle not hypertrophied.

The vessels are greatly thickened, embedded in large quantities of fibroid material (see Pl. XVI). This applies to arterioles and capillaries. This fibroid thickening is seen extending in great quantities from the adventitia of the vessels into the surrounding connective-tissue plexus, clearly denoting that the vessels have been centres of thickening, and that the fibroid change has radiated from them (see Pl. XIX). These appearances lead us to infer that the fibroid change originated in the posterior and lateral columns around vessels and along septula, and that they were chronic in character. Many nerve-tubules were atrophied or contracted by the growth; but there was evidence also showing that acute changes had supervened on the chronic, swelling and destroying many tubules which had escaped the fibroid thickening.

In the cord we have last to mention (M—[1]) the fibroid changes are extremely marked, and in the posterior columns most of all. A portion of the surface of the posterior column is seen to be much puckered, as if drawn inwards by fibroid contraction. One of the most striking features of this cord is an extraordinary thickening of the vessels; their intima is sharply bounded; outside that is a clearer stratum, and that again is bounded by the coarse fibres of the adventitia, and from it radiates a felt-like fibroid material studded with nuclei and granules (see Pl. XVII). As many as thirteen of these thickened vessels are counted in the field under Hartnack's objective No. 7, ocular No. 3, but it is very difficult to determine the condition of much of the surrounding connective tissue; it is studded with granular matter and nuclei; it looks as if it had undergone general fibroid change. There appear to be many shrunken axis-cylinders; others look large, either swelled or hypertrophied.

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[1] Case 12, of Stephen M—, æt. 52. Autopsy showed fibroid consolidation of upper lobes of lungs; heart, no noticeable change, except some dilatation of right side; fibroid thickening in liver; testicle smaller than normal, tough, firm, and invaded by fibroid material; kidneys normal; spleen normal.

It is a very striking feature in this cord also, that both the connective tissue and the nerve-tubules seem comparatively little affected in some portions of it. But even where the changes are in comparison very slight, the fibrils are in parts thickened by fibroid material, and scattered amongst it are many very small nuclei, and the vessels are thickened by fibroid substance. This fibroid change is seen extending from the surface in many parts. In one part a broad wedge-shaped fibroid mass is traced from the surface inwards right across the field (see Pl. XVII). Many nuclear bodies are embedded in the fibroid substance. As it tapers off, thick vessels are observed lying in it. At its summit it bifurcates; one extremity includes three thickened vessels; the other seemingly spreads out, embraces vessels, and a network of new connective tissue, consisting of nuclei and fine fibrils, extends from around the vessel. This new tissue together forms a broad mass with no recognisable nerve-structure in it, and occupies about a third of the field. Between the two arms just named, a group of nerve-tubules is seen, with their axis-cylinders and medullary sheaths little altered. There is so much morbid change in this cord that we can here only describe some of the most prominent features.

In some of the cords described there was a large quantity of dense protoplasm accumulated in and around the vessel walls, causing great thickening. This material differed from the simpler exudation by its greater density (see Pl. XVIII) and less hyaline character; by taking dye more readily, and by the increased nuclear bodies in it. These changes seemed to us to denote myelitis more or less acute.

In other cords the arterioles and capillaries were surrounded by large quantities of felt-like fibroid material (see Pl. XIX), which extended into and between the nerve-tubules. Where the connective tissue was much thickened, one or more vessels were commonly seen embedded in it. This fibroid thickening corresponds to what Charcot, Leyden, and others, have named and described as sclerosis, and to what Rindfleisch calls "inflammatory induration." Rindfleisch says, "An attentive examination of the smallest of these foci leads to the curious discovery that the masses of fibroid thickening have all got a red spot or line in their centre, a distended blood-vessel. . . . . All these vessels with their finer ramifications are in a state which we should not scruple elsewhere to call one of chronic inflammation."

He particularly refers to the increased cells thickening the adventitia of the arterioles and capillaries, which, as we also have stated, is seen more especially in the acuter changes, and he goes on to remark, "In these alterations of individual vascular tufts I see the first anatomical element of the disease; the second consists in a fibroid metamorphosis and overgrowth of the neuroglia."

Our observations on the cord show that exudations from the vessels lead to swelling and thickening of its perivascular and other tissues. This is acute in some cases; in others the change is much more chronic. Not only is there in the acute cases the traceable exudation from vessels evidently causing the surrounding thickening, but in the chronic cases also the large collection of fibroid material around the arterioles and capillaries, becoming thinner and thinner as it recedes from them, supports the opinion that the fibroid change begins in and around the walls of the arterioles and capillaries.

We have not yet, however, arrived at any conclusion as to whether there is merely an excessive discharge of blood-plasma, or whether the plasma accumulates because the lymphatics are blocked, or the connective-tissue cells or areolæ thickened, blocking and preventing the plasma passing onwards into the lymphatics. In whatever way it happens the plasma accumulates most probably first around the capillaries, then in the capillary wall, and as the obstruction there is increased, the tension is transmitted backwards, and exudation into and around the arteriole wall follows as a consequence. In support of this statement Rindfleisch notices, as we have done, that the calibre of the affected arterioles seems increased.

Having thus described the changes we have observed in these cords, it remains to summarise our conclusions.

1. In two cases (H— and B—) with granular and contracted kidney there were no appreciable changes in the cord; in two others (D—e and D—n) the arterioles and capillaries of the cord were hyaline and much thickened. [The details of these cases are here not introduced.]

2. In one case (L—), in which there were granular contracted kidneys (with indications of recent acute nephritis) and well-marked hypertrophied heart, there was seemingly exudation into the cord substance (œdema).

3. In two cases (W— and C—) in which there were the usual changes of chronic Bright's disease with contracted kidney (heart hypertrophied, kidneys contracted), some arterioles and capillaries of the cord were thickened by fibroid material, with or without hyaline appearance; others were swelled and hyaline only. The connective tissue in parts looked rigid, and there was exudation material around and into it and the nerve-tubules—fibroid change and œdema.

4. In another case (W—) no traces of chronic fibroid changes were found in the arterioles and capillaries, but their walls were greatly swelled by exudation of homogeneous hyaline material, which extended in broad tracts from them into the surrounding tissues (initial myelitis ? hypostatic ?).

5. In three cases (H—, F—, and B—) some of the arterioles and capillaries were much thickened, their walls swelled by hyaline material, and without any or but doubtful evidence of chronic fibroid changes in them. The connective tissue was swelled and the fibrillar character lost, seemingly in consequence of large exudation of hyaline homogeneous material into it. Its nuclei were multiplied (myelitis). But whilst the condition of some of the arterioles and capillaries and the connective tissue and nuclei was thus changed,

there were other arterioles apparently thickened by coarse fibroid material, indicating more chronic changes preceding the acute.

6. In two cases (K— and C—) arterioles and capillaries were here and there thickened by fibroid changes, and there were numerous centres of thickening of connective tissue with atrophy or contraction of nerve-tubules, whilst in many other parts the cord substance looked strikingly healthy (*sclérose en plaques*). In one of these cases (C—) the brain was atrophied; the heart was greatly hypertrophied, weight twenty-two ounces, valves healthy; kidneys venously congested, and with indications of only a little recent suppurative nephritis, excited by the paraplegic cystitis. In the other case (K—) the kidneys were granular and contracted; heart hypertrophied; no valvular disease.

7. In three cases (S—, P—, and M—) the arterioles and capillaries were much thickened by fibroid material, and sections of these vessels showed them embedded in large quantities of felt-like fibroid substance, which extended from them, dividing and subdividing, and invading and destroying medullary sheaths and axis-cylinders, or enclosing other nerve-tubules in a coarse felt-like connective tissue. Near the exit of the posterior nerve-roots from the surface of these cords the connective-tissue thickening was especially great, and many nerve-tubules at this part were replaced by fibroid material (diffuse sclerosis).

It may be superfluous to repeat that, notwithstanding the destructive changes we have described in these cords, a considerable portion of their structure remained comparatively healthy, and that fact elucidates some of the peculiarities of the clinical features observed in these cases. From the detailed particulars of the arterioles and capillaries it may be inferred that the walls of some were simply swelled up by hyaline albuminoid material, without much increase of the nuclei of the intima or adventitia in number; and in referring the changes observed in the cords of L— and W— to simple exudation, we are unable, from the appearance of the cord and exuded material, to determine to what extent it occurred shortly before death, during dying, or even after death. In the case of L— there was general œdema in connection with acute nephritis; the exudation into the cord-substance might therefore be part of the general serous accumulation. In the case of W— there was no renal disease and no general œdema.

The hyaline material had, in W—'s case, spread widely into the textures of the cord; here and there stream-like extensions disintegrating the cord-substance. The connective-tissue nuclei seemed altered, but it might fairly be questioned if they were increased in number. These appearances seemed to us to indicate that the exudation had most probably occurred during the venous congestion of dying, and may be even, to some extent, after death. We still think it most probable such was their origin. We have to qualify this, however, by stating: (*a*) That we have not found a similar amount of exudation of hyaline material in healthy cords of persons killed, but in a minor degree we have found this hyaline swelled appearance in the connective tissue of the cervical cord of a man killed by fractured skull and lacerated brain (B—). It was instructive to notice that then the appearance was less marked in the dorsal cord, leading to the inference that the exudation and swelling were due to the injury to the head. (*b*) That we have not found similar exudation either in amount or manner of arrangement in most cords diseased. (*c*) That we have seen similar exudation, but in less degree, in the cord of a person who died of tetanus. Here the vessels of the cord were very full of blood, and their walls were hyaline, and the connective tissue also.

We are led by these facts to reserve the question of the origin of this simple exudation for further examination.

The fibroid changes observed in several of the cords described resemble those of granular contracted kidney in the following features:

In the spinal cord, as in the kidney, the fibroid change, as might be expected, is most marked where the connective tissue is most abundant. It extends in the cord, as in the kidney, from the surface-membrane inwards; or in the cord from the grey matter outwards, and in the kidney from the base of the cones inwards: and in both cord and kidney it extends from the adventitia of the arterioles and capillaries into surrounding connective tissue. The fibroid material in the cord, as in the kidney, contracts and compresses surrounding tubules, atrophying or destroying them, but leaves many other adjacent tubules comparatively normal. In the cord, as in the kidney, it would seem that acute change commonly supervenes on the chronic. Seeing that so many tubules remain comparatively normal, we are enabled to understand how it is that both cord and kidney may retain much of their functional activity, even when they are the seat of very extensive fibroid change, and this usually continues (persons walk or secrete urine fairly well) until the more healthy tubules are deteriorated by acute changes.

It only remains to state that of the five cases in which there were well-marked fibroid changes (sclerosis) in the cord, in two (S— and K—) the kidneys were very granular and contracted, left ventricle of the heart hypertrophied, no valvular disease.

The kidneys were slightly granular, and the left ventricle of the heart not hypertrophied, in one (P—).

The kidneys were not contracted nor granular in two; but in one of these (M—) the lungs were the seat of extensive fibroid induration and the testicle also, and in the liver a little similar change; in the other (C—) the left ventricle of the heart, without valvular disease, was greatly hypertrophied, and the brain atrophied.

These particulars show that fibrosis in the cord may occur coincidently with fibrosis of the kidney; or it may be in advance of the fibroid change in the kidney; or occur as part of a general fibrosis, altogether independently of renal disease.

We cannot conclude these observations without expressing our obligations to Mr. Robert Kershaw for the great care, patience, and skill with which he has prepared the sections; to Dr. Turner, for much help in collecting and revising details; and to Mr. Hollick, the artist, for the extreme care with which the drawings have been executed.

Note.—In the 'Pathological Society's Transactions,' vol. xxvii, 1877, a paper criticising the theory of arterio-capillary fibrosis, appeared, entitled "On the Changes in the Blood-vessels and in the Kidney, in connection with the Small Red Granular Kidney," by George Johnson, M.D.—(Ed.)

DESCRIPTION OF PLATE VII.

Plates VII to XIX inclusive illustrate the Observations of Sir William Gull and Dr. Sutton on the Changes in the Spinal Cord and its Vessels in Arterio-capillary Fibrosis. (Pages 391—414.) The original drawings were made by Mr. A. T. Hollick.

Plate VII.—Drawings represent sections of a healthy cord, from a boy, æt. about 14, killed by an accident.

Fig. 1. From the lateral column near the surface and posterior nerve-root, showing the fibrillar character of the connective tissue, radiating from centres, dividing and subdividing; its finest branches surrounding the individual nerve-tubules in the form of a plexus. In each of these centres, where a nucleus is usually seen, there is a quantity of granular matter, but the nuclei themselves are not represented.

α. Faint hyaline cloudiness (albuminoid material?) seen pervading the nerve-tubules in some parts.

Fig. 2. From the lateral column, near the grey matter, showing—

α α. Vessels (finest arterioles) cut longitudinally and transversely, from the outer coat of which the connective-tissue fibrils extend between the nerve-tubules.

β. A centre from which the exceedingly fine connective-tissue fibrils are radiating, and in which a nucleus is distinctly seen.

Several other similar centres with the nuclei are represented in the drawing.

Plate VII.

Fig 1.

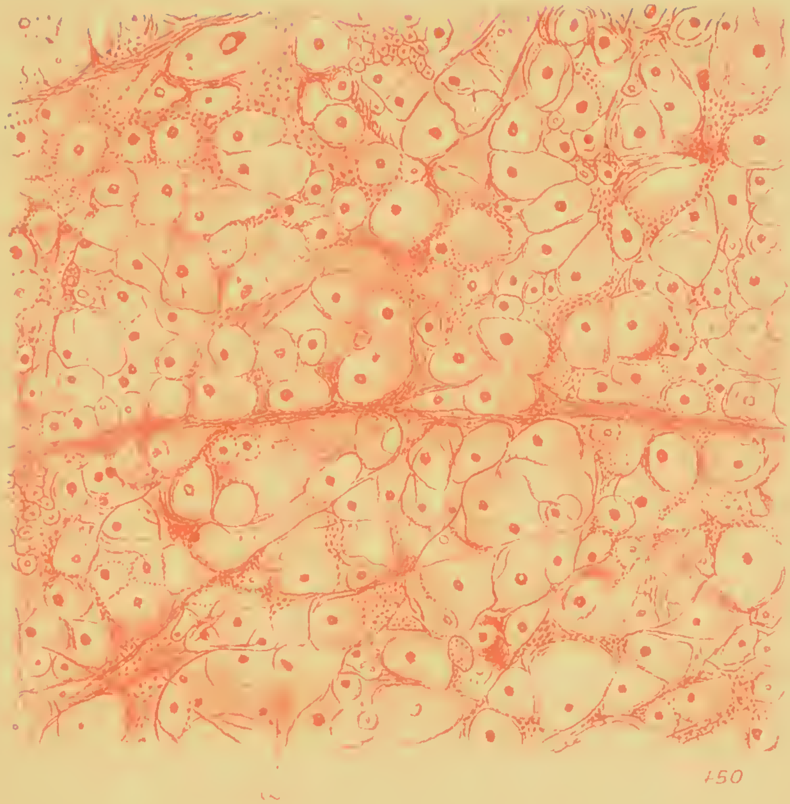
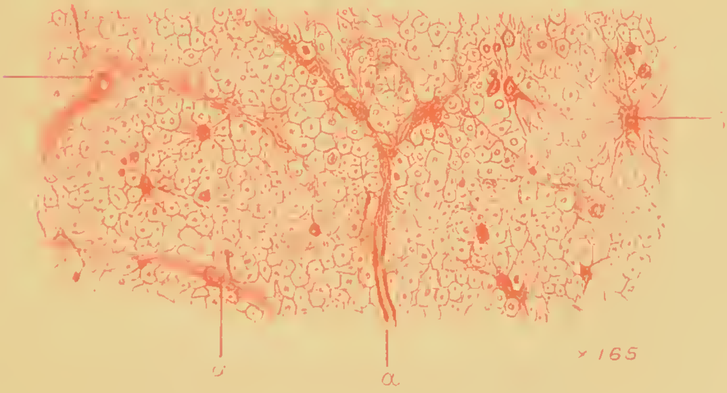


Fig 2.



West, Newman chromo.

DESCRIPTION OF PLATE VIII.

Drawings represent sections of the healthy cord of a man, æt. 20, who was killed by an accident. Intended to show the appearance of the connective-tissue cells and their radiating fibrils. [The artist has drawn the fibrils too coarsely.]

FIG. 1. From the lateral column, close to the posterior cornu, showing—

α. The floor of a vessel cut longitudinally, its outer sheath remaining, from which fibrils of connective tissues are given off.

β β. Connective-tissue nuclei, with a small quantity of surrounding protoplasm, from which fibrils radiate, dividing and subdividing, enclosing the nerve-tubules, and forming the connective-tissue plexus.

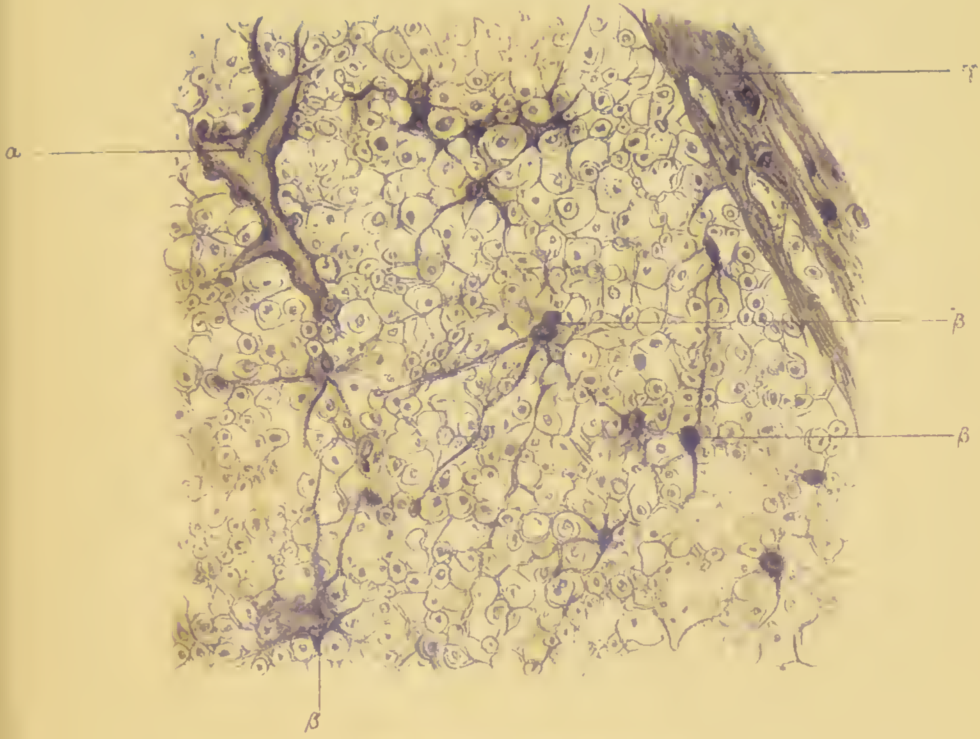
γ. Grey matter of the posterior cornu. (× 330.)

FIG. 2. From the same region of the cord.

α. An arteriole cut longitudinally, coloured corpuscles occupying its lumen. The small amount of perivascular connective tissue round it is to be noticed. (× 250.)

Plate VIII.

Fig 1.



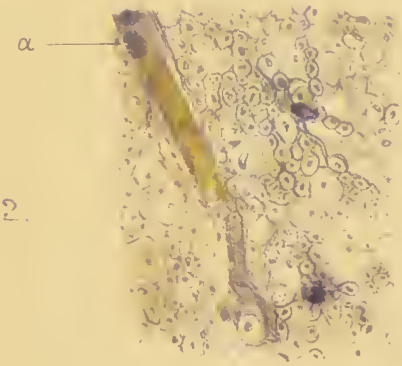


Fig 2.

T. Hollick del

West, Newman chromo.

DESCRIPTION OF PLATE IX.

Drawings represent sections of the cord of a man, æt. 25 (L—), who died with granular contracted kidneys, mottled by acute nephritis; hypertrophy, with dilatation of the left ventricle. Clinically there was œdema, albuminuria, and indications of "uræmia," &c., but no symptoms of spinal cord disease are recorded.

FIG. 1. From the lateral column in the dorsal region, near the surface and the posterior nerve-root.

α α. Swollen axis-cylinders.

γ. Granular matter along the plexus of connective tissue, thickening it.

FIG. 2. Another part of the same specimen.

β β. "Colloid masses," so called, hyaline, homogeneous, (albuminoid?) material.

γ. Granular matter along the connective-tissue plexus, thickening and obscuring it.

These appearances are considered to be due to œdema of the cord.

Plate IX

Fig 1

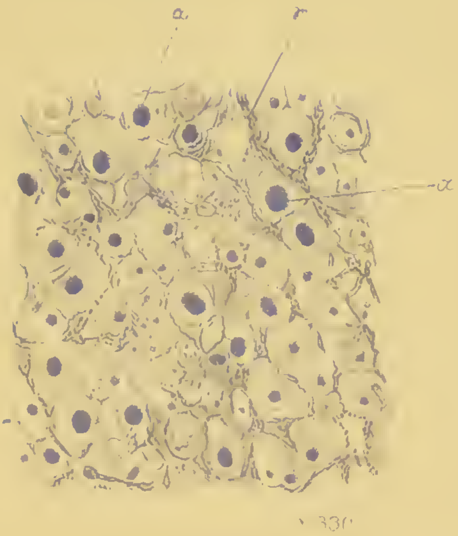
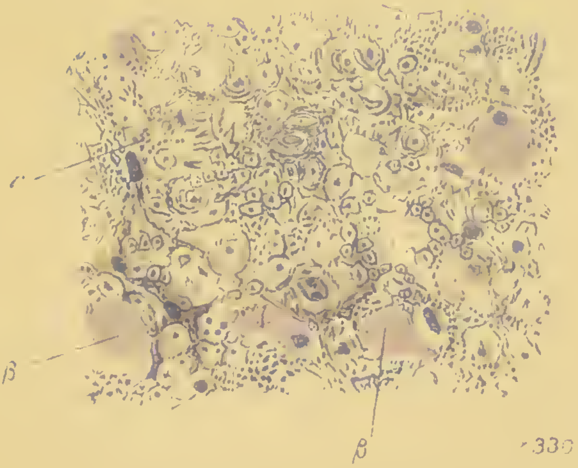


Fig 2



A. F. Hollick del.

West, Newman chromo.

DESCRIPTION OF PLATE X.

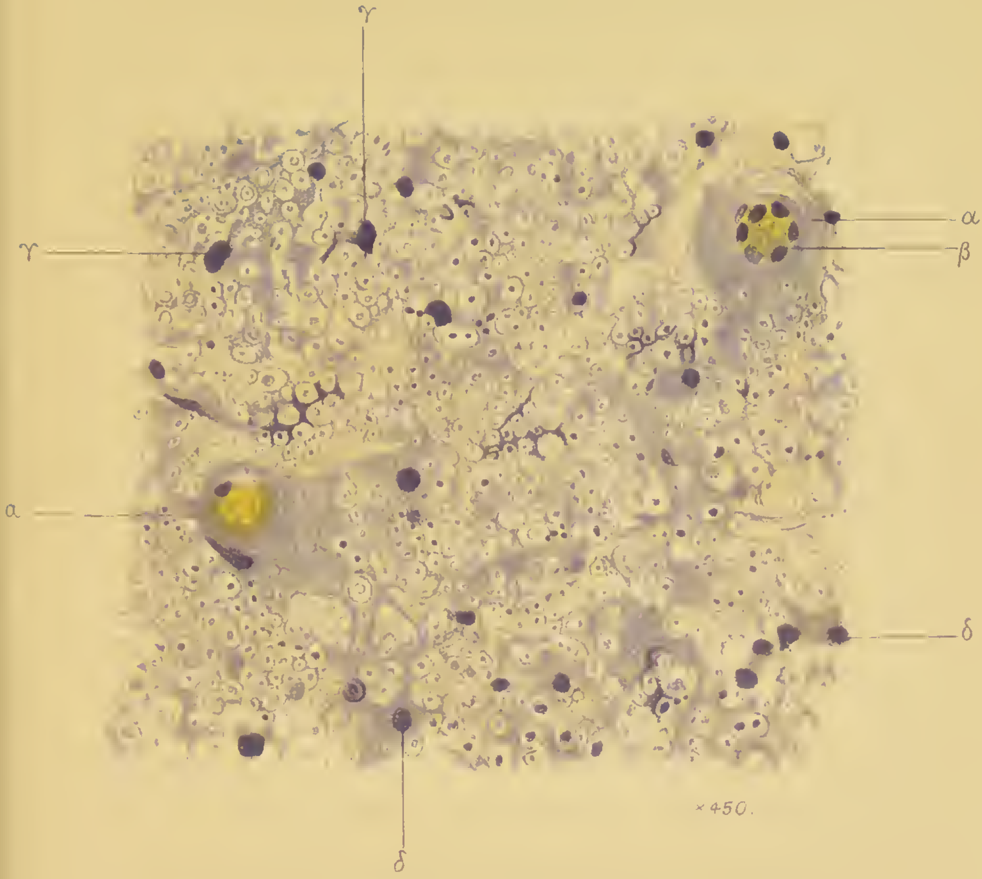
Drawing represents a section of the cord (lumbar region) of a man, æt. 47 (B—), who died from pneumonia and pericarditis, with granular contracted kidneys and hypertrophy, with dilatation of the left ventricle of heart. The cord-substance, on section, looked wet—œdematous. No symptoms of cord disease noted. The patient walked into the hospital, but became comatose shortly afterwards.

The drawing shows arterioles much thickened, with the nuclei of their "intima" multiplied; and exudation material, which, in escaping, has swelled and clouded the vessel wall, and extending from it along the connective-tissue fibrils, has swelled and disfigured them also.

a a. Vessels swelled by exudation.
β. Nuclei of intima swollen and multiplied.
γ γ. Swollen and deformed connective-tissue nuclei.
δ δ. Leucocytes, or connective-tissue nuclei.

This drawing is considered to represent commencing myelitis.

Plate X.



[illegible] del

West, Newman chromo

DESCRIPTION OF PLATE XI.

Drawing represents a section from the dorsal region of the same cord (B—), but showing a more advanced change in myelitis.

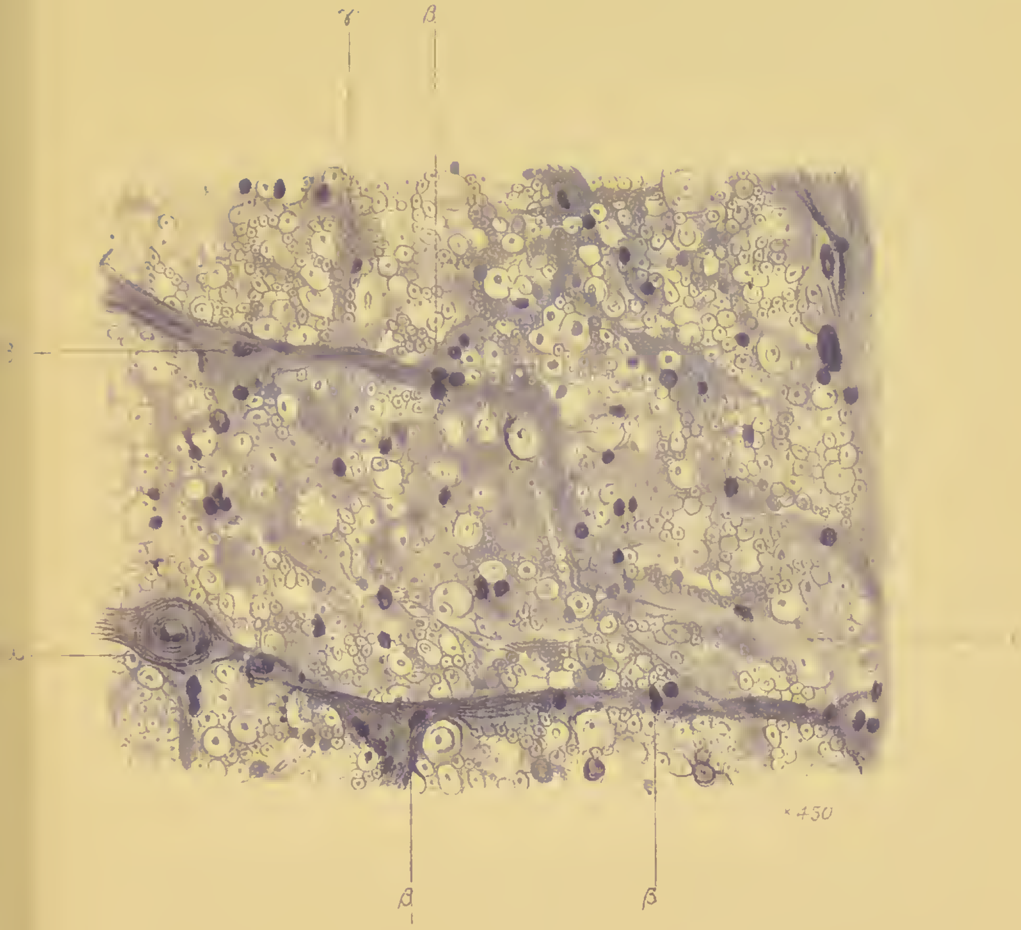
a. Thickened vessel, hyaline layer.

β β. Thickened connective tissue; the nuclei multiplied, grouped in twos and threes.

γ γ. Nerve-tubules invaded (atrophied?).

There were also fibroid appearances in this section, but to what degree the changes were old we could not determine, because much of the tissue was evidently swelled and obscured by recent exudation.

PLATE XI.



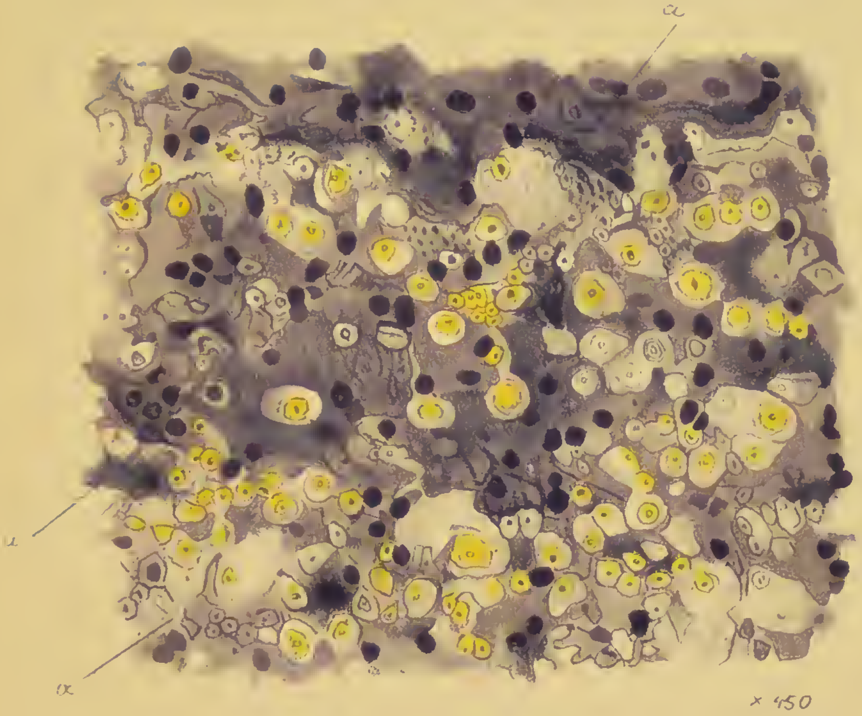
[illegible] del. West Newman chromo.

DESCRIPTION OF PLATE XII.

Drawing represents a section taken from the lumbar region of the cord of a man, æt. 36 (G—), who was admitted with symptoms of loss of co-ordination of arms, legs, chest, and articulation. Left ventricle was hypertrophied and dilated, no valvular incompetency. No disease of the kidneys; they were simply congested. The posterior cornua of spinal cord were observed to be indistinctly defined, and the posterior columns had a decided abnormal appearance.

The drawing is considered to show still more advanced changes (subacute myelitis?) in the cord. In the previous plate the nuclei are seen for the most part small and together, as if dividing; here they are more generally separated and multiplied. With the increasing nuclei large accumulations of dense protoplasm are seen (*a a*) of a more formed appearance, and more extensively invading and obscuring the nerve-tubules.

Plate XII



A. T. [illegible] del.

West Newman chromo

DESCRIPTION OF PLATE XIII.

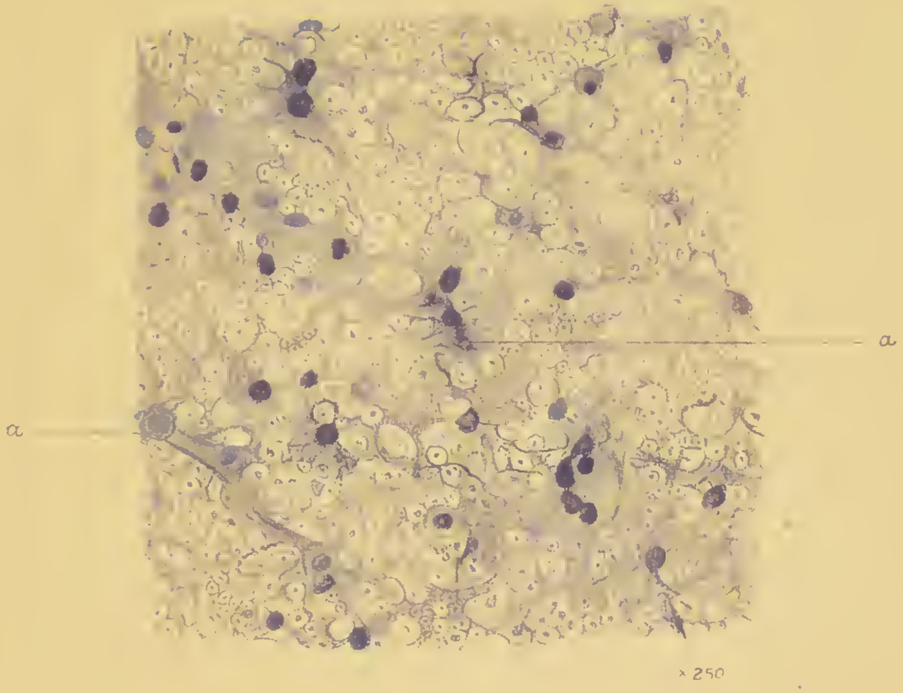
Drawing represents section of the cord of a man, æt. 43 (K—), who died with granular contracted kidneys and hypertrophied and dilated left ventricle. The clinical cord symptoms in this case were those of progressive muscular atrophy and paralysis of hands and feet.

The appearances represented are those corresponding to "*sclérose en plaques.*"

a a. Fibroid thickening around the connective-tissue nuclei, broad strands extending from them, contracting and constricting the nerve-tubules.

[The felt-like appearance of the fibroid material is not sufficiently shown in the drawing.]

Plate XIII.



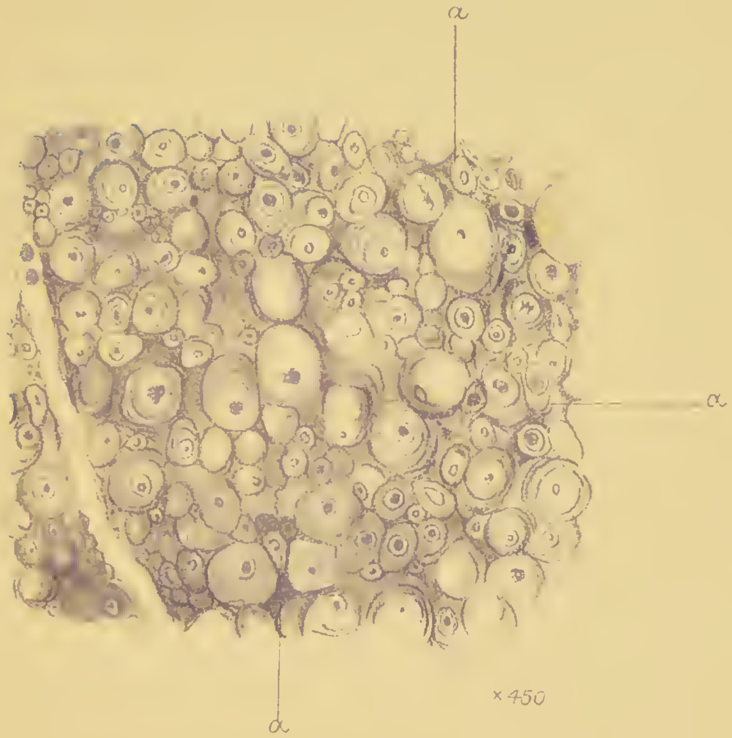
[illegible] del. West, Newman chromo.

DESCRIPTION OF PLATE XIV.

Drawing from the cord of a man, æt. 40 (S—), whose spinal symptoms, as in the last case, were those of progressive muscular atrophy, and who died with hypertrophied and dilated left ventricle, and with kidneys granular and contracted, and mottled by acute nephritis.

The drawing shows still further advanced changes in the connective tissue of the cord. The nuclei have mostly disappeared. The intertubular changes (*a a*) are not so much in amount as in the former plate, but are more rigid in character.

Plate XIV.



A T Hollick del.

West, Newman chromo

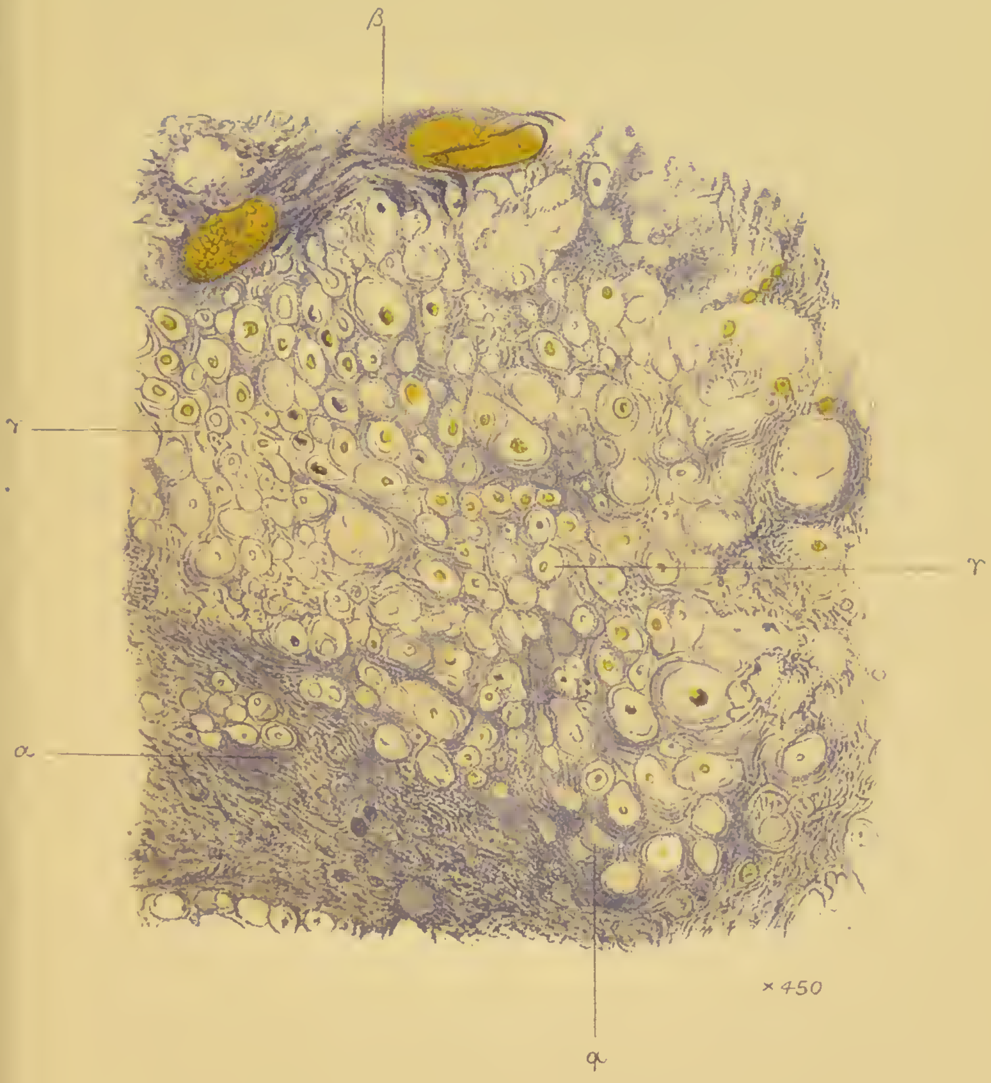
DESCRIPTION OF PLATE XV.

Drawing from the same case as the last plate, representing a section of the lateral column, close to the surface and near the posterior nerve-root.

The appearances shown are those of extreme sclerosis.

- *α α.* Masses and broad tracts of felt-like fibroid material invading and contracting the nerve-tubules.
- *β.* Fibroid thickening around a vessel; its lumen is occupied by coloured blood-corpuscles.
- *γ γ.* Various gradations of contraction and destruction of the nerve-tubules.

Plate XV.



A T Hollick del

West, Newman chromo

DESCRIPTION OF PLATE XVI.

Drawing represents section from the cord of a man, æt. 58 (P—), with symptoms indicating acute softening of cord supervening on slow failure of motor power in legs, accompanied by pains and twitching in the legs. At the autopsy the cord appeared normal (?) ; the heart healthy; kidneys slightly granular; lungs emphysematous.

The part shown is in the lateral column, near the posterior nerve-root, and close to the surface, presenting acute changes upon extreme sclerosis.

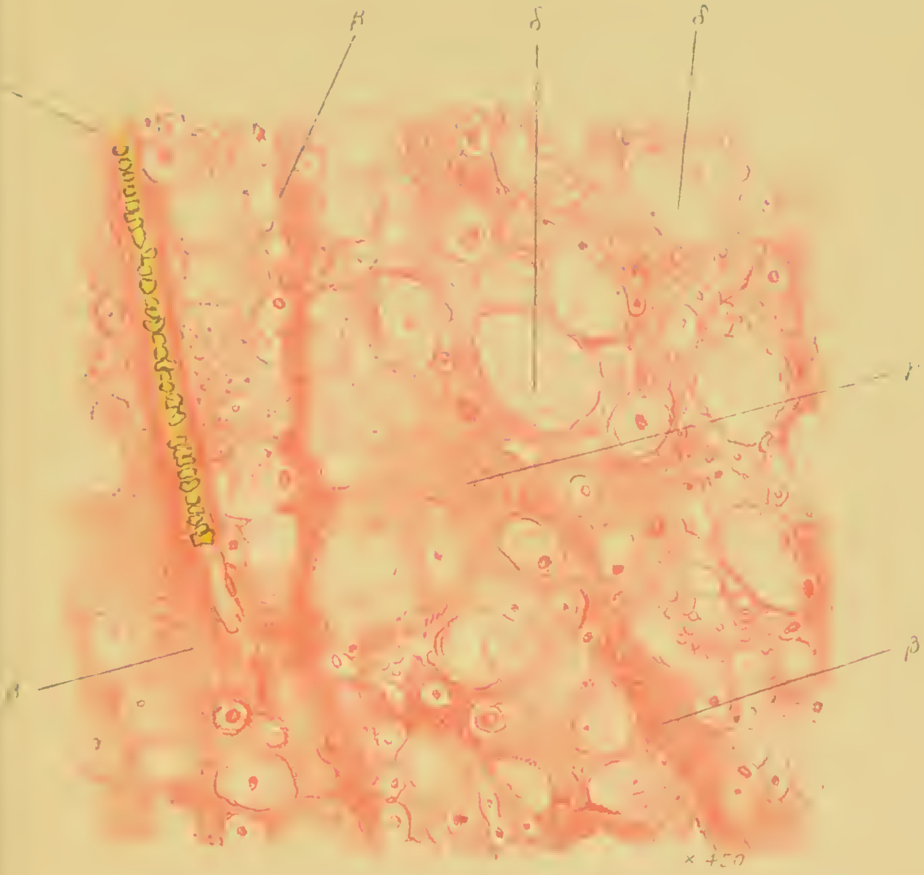
α. A vessel thickened by fibroid material.

β β. Extreme fibroid, felt-like thickening, which has invaded and destroyed the nerve-tubules.

γ. Destruction of the nerve-tubules in many parts.

δ δ. Nerve-tubules swollen and seemingly disorganised (acute softening ?).

Plate XVI.



T Hillier del.   West Newman chromo.

DESCRIPTION OF PLATE XVII.

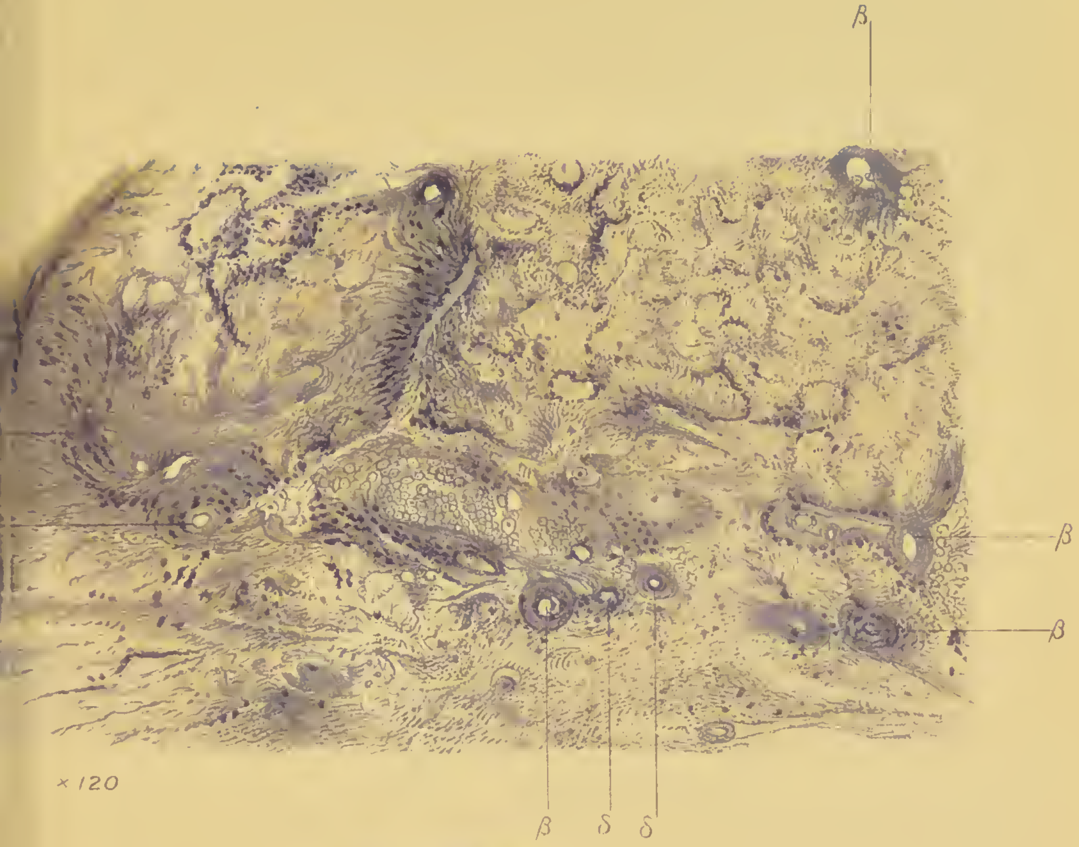
Drawing represents section from the cord of a man, æt. 52 (M—), a paraplegic patient, who had also paralysis of the sphincters. After death extensive fibroid consolidation of the lungs was found and fibroid degeneration of the testes. The cord was soft in the upper dorsal region, but this was attributed to crushing in taking it out of the body. The heart and kidneys were normal. There was a history of syphilis thirty years previously, and of a fall on the back seventeen years before death, followed in a short time by incomplete and temporary paraplegic symptoms in arms and legs. He recovered, and followed his employment for seventeen years, and then the fatal paraplegia supervened.

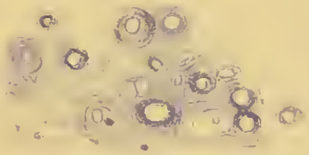
In the larger drawing a broad, wedge-shaped tract of dense fibroid thickening is seen extending inwards from the surface, and other tracts of similar tissue around the vessels in the posterior column.

α. Region where the nerve-tubules have been destroyed.
β β. Arterioles greatly thickened.
δ δ. Capillaries thickened.

The smaller drawing represents a group of thickened arterioles, and capillaries from a part nearer to the grey matter.

Plate XVII.





P. Hall ck. del

West, Newman chromo.

DESCRIPTION OF PLATE XVIII.

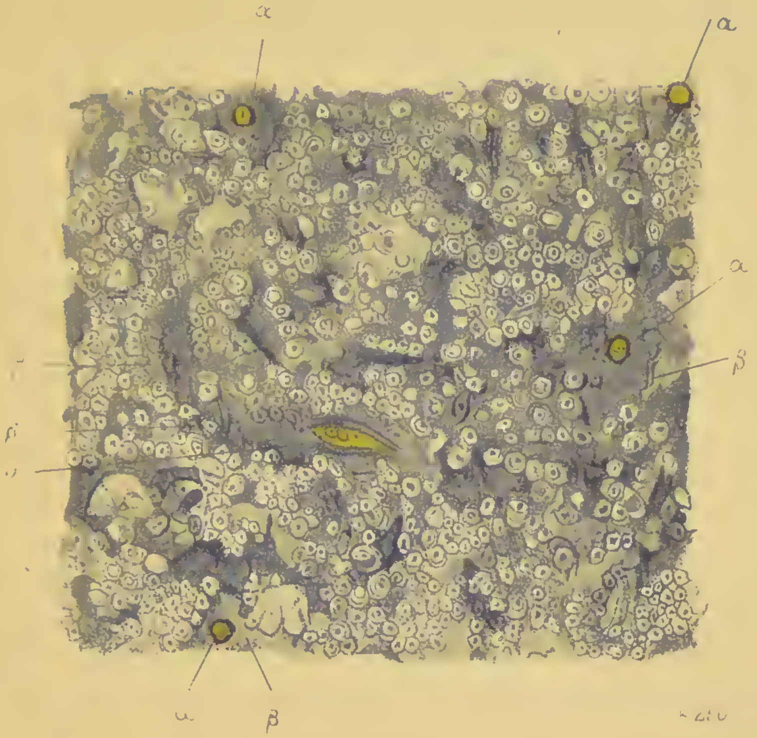
From the same case as Plate XII (G—). The drawing is intended to show the great accumulation of dense protoplasm surrounding the arterioles and capillaries (*a a*), and extending from them along the tracts of the connective tissue, thickening it.

The specimen is stained with aniline black, which does not show the connective-tissue nuclei, which are conspicuous in the former plate referred to.

αα. Arterioles and capillaries filled with coloured blood-corpuscles.

ββ. Dense masses of protoplasm thickening the vessels and extending from them.

Plate XVIII



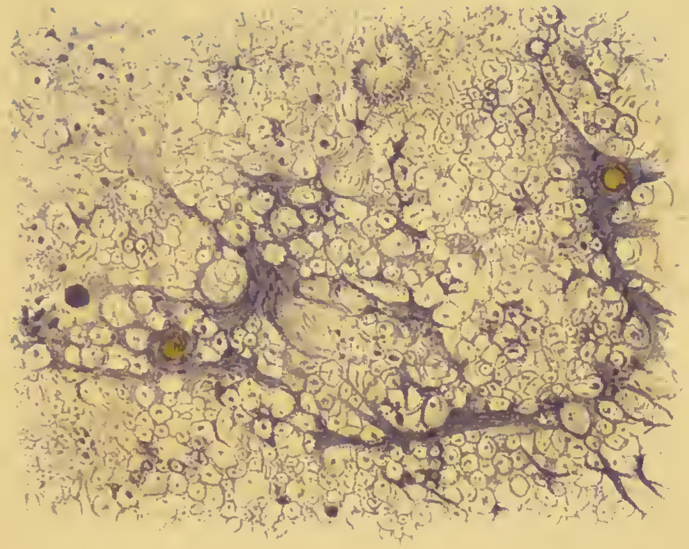
Hollick fe.

West Newman chr.

DESCRIPTION OF PLATE XIX.

From the same case as Plate XVI. Fibroid, rigid, felt-like material is seen surrounding the arterioles, thickening their walls, and extending from them in tracts along the course of the connective-tissue fibrils. Plate XVIII shows that dense protoplasm collects and thickens the vessel walls, and that the thickening radiates from the arterioles and capillaries in the course of the connective tissue. Here is shown (in Plate XIX) a more advanced change than that represented in previous plate, a stage of sclerotic contraction beginning around the vessels. In other parts of this section the contraction is extremely advanced (see Plate XVI).

Plate XIX



A. T. Hollick del. West, Newman chromo

DISCUSSION ON THE RELATION

OF

RENAL DISEASE TO DISTURBANCES OF THE GENERAL CIRCULATION,

AND TO

ALTERATIONS IN THE HEART AND BLOOD-VESSELS.[1]

The above thesis does not obviously raise the question of names as to what is or what is not Bright's disease. It does, however, challenge the long-prevalent views that the disturbances in the heart and general circulation occurring with renal disease are due to it. Of course there are to be eliminated all the cases of primary valvular and similar mechanical conditions of the circulatory symptoms, generally admitted to have an origin quite independent of kidney disease, and leaving on one side cures of accidental albuminuria which thus arise.

Before proceeding to read the few condensed remarks we have put together, we will put in the forefront the abstract of our views, viz.—

1. Kidney disease is associated with or causes changes in the circulation, heart, and blood-vessels variously, according to the kind and seat of the morbid changes in the renal

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[1] By Sir William Gull, Bart., M.D., F.R.S., and H. G. Sutton, M.B. Reprinted from the 'Transactions of the International Medical Congress,' London, 1881, vol. i, p. 374.

tissues, *e. g.* vascular (arterial or venous) or tubular or mixed (parenchymatous nephritis).

2. Kidney disease may be dependent upon causes primarily weakening the circulation, *e. g.* causes of general malnutrition, phthisis, fever, scrofulosis, alcoholism, syphilis, &c.

3. Defective renal function has a weakening influence on the circulation and nutrition; tissues become choked by œdema, enfeebled by anæmia and uræmia, and generally wasted, *e. g.* mottled or large white kidney, surgical kidney, &c.

4. Kidney disease may be dependent upon causes primarily leading to thickening of heart and blood-vessels generally, and to obstruction of the interstitial circulation through the several tissues, *e. g.* arterio-capillary fibrosis, climacteric changes.

5. The question as to the effects of kidney disease on the circulation may often with advantage be reversed, namely, as to what is the influence of alterations in the circulation in producing kidney disease: *e. g.* abnormal venous tension, arterial tension.

6. Many of the changes in organs, hitherto considered uræmic, are referable to tissue changes, capillary and interstitial, atrophic, anæmic, effusive, fibroid, &c., and may be independent of defective renal excretion.

Kidney *function* can only be understood through the physiology of the general circulation; kidney *disease* can only be known through the disturbances in the general circulation.

Clinical and anatomical facts show that the disturbance in the general circulation associated with, or dependent on, kidney disease, varies according to the kind of morbid change in the kidney; for instance, the disturbance in cases of suppuration of the kidney, (1) "surgical kidney," is altogether different from the disturbance in cases of (2) "large white kidneys" or (3) granular contracted kidneys.

The antecedent conditions of kidney disease, as in cases of scrofulous pyelitis, lardaceous disease of kidney, or large white kidneys, cause of themselves disturbance in the general circulation—as, for instance, in phthisis, syphilis, abuse of alcohol, fevers, heart disease, &c.; and when from these the kidney disease supervenes and increases there is *additional* disturbance from that source in the general circulation. How much of the whole disturbance is due to the primary causes, and how much to the secondary kidney disease itself, is an exceedingly difficult problem; the actions and reactions may be considered until the answer is in many cases reduced to this paradox, *that the disease is local because it is general, and general because it is local.*

Kidney disease may be grouped into tubular, vascular, and mixed forms; in mixed, both tubular and vascular structures are about equally affected. This latter is the common form.

I.—The tubular, in its most traceable form, is seen in "surgical kidney," or in strumous pyelitis. The morbid changes extending from the pelvis along the tubules to the cortex.

There is retained excretory matter, and a pathological experiment is thus performed, showing the effect of such retention on the general circulation.

There is often ultimate decomposition in the organic matters of the excreta into ammonia and carbonic acid, and an extreme poisoning of the system.

It is thus clear what kind of disturbance is produced in the general circulation, when excretory matters are poisonously retained. There is not œdema, nor symptoms of uræmia, so called; little or no vomiting, little or no dyspnœa, and no cardiac hypertrophy. There is increasing failure of nervous and muscular energy; the pulse and heart become feebler, mucous membranes become hyperæmic and catarrhal, consciousness remains clear, and there is yellowness of skin, denoting change in blood-corpuscles.

II.—In cases of "intratubular nephritis," glomerular or other, there must of course be disturbance in the vascular system of kidney congestions and exudations, and in many cases more or less fibroid intertubular change, so that these are often inseparable anatomically from the mixed group.

But it is useful here to consider the intratubular cases in their simpler form, without fibrosis and without cardiac hypertrophy, so as to estimate what disturbance occurs in the general circulation when the *water function* of the kidney fails.

These cases are characterised by great disturbance both in the general vascular and general interstitial circulations.

Speaking generally, in these intratubular cases, the urine, at the outset of the disease especially, is diminished day by day, but weeks usually thus elapse before there is any marked œdema. The skin after awhile becomes puffy, the breath short, the nervous system much disordered, &c.

Failure of water function of kidney does not cause marked œdema, until the skin and lung water functions fail also.

It is an induction from clinical and anatomical facts, that as the outflow of water from the kidneys lessens, *cæteris paribus*, the water increasingly accumulates in the interstitial tissues, lymphatic spaces, and serous cavities: with this increased hydræmia the arterial tension is raised, and there is increased tension in skin and lungs; breathing becomes more difficult, skin becomes choked with serous fluid (œdema), and ultimately air cells and bronchi filled with serum (œdema of lungs), or may be of pleura.

As aspiration of chest (inspiration) lessens, the venous circulation is obstructed and the right heart over-distended. As the lungs are choked by serous fluid, the air circulation in blood and interstitial tissues is hindered, less air being inspired, the water (blood) circulation diminishes and gradually ceases, since the evaporation of air from the venous blood is itself a factor in the blood movement. For it cannot be overlooked that gases in a fluid being less affected by gravitation, and their particles, especially under the circumstances, being more prone to separate from those of the fluid itself in which they are contained, they would concur with the circulating force as against the inertia of the fluid itself. Anæmia supervenes and increases, indicating that oxidation is diminished, and that less heat is produced. It is here to be remarked that air being much lighter than water, it is more easily responsive to heat than water; more quickly expanded and compressed; whilst water, being more easily controllable and incompressible, dissipates energy less quickly; it is adapted to store more; it may consequently be inferred, and it is demonstrable, that in the general circulation there is an auxiliary action and mutual dependence between these two mediums, the water and the air circulation, and that as one fails, the other fails.

An increasing anæmia is the worst sign; it reveals that heat and light energy have greatly diminished in the circulation, and that the normal *protoplastic operations in the production of the blood-corpuscles are failing.*

The sense organs, lungs, red corpuscles, and locomotive organs are, in the course of animal life, correlatively and proportionately developed ; and they correlatively and proportionately fail, as in phthisis and Bright's disease.

With the failure of the water and of the air circulation, there is disturbance in the nervous system ; partly due to œdema and anæmia of sense organs ; skin, retina, spinal cord, brain, &c.

It can be shown that there is local disturbance in the interstitial circulation of cerebro-spinal system, coincident at least with the so-called uræmic symptoms ; albumenoid and corpuscular exudations, swelling of nerve tissues, &c.

It is common experience also that the tissues—mucous membranes and others—swell through œdema, and become additionally swollen by inflammatory exudations ; and life is thus ended by pericarditis, peritonitis, pneumonia, myelitis, &c.

Taking a wide survey, it is clear that the special and the general functions fail coincidently ; that as the special water function of kidney, lung, and skin fails, the water function of the general protoplasm (connective tissues, capillary walls, &c.) fails also ; that as the air function of lung fails, the air circulation of interstitial tissues fails also.

Of these cases of intratubular nephritis, it is here to be noticed that their antecedents, viz., phthisis, heart disease, abuse of alcohol, syphilis, &c., bear witness in themselves that there is primary failure of nutrition throughout the body in the protoplastic circulation ; and that the interstitial excretory function fails before the special (renal).

The cardiac changes are those of dilatation of right ventricle, or chiefly. They may, however, not be more than is answerable to the impeded circulation from the causes here detailed.

The vascular form of kidney disease is witnessed in the granular contracted kidney, and in many such cases it is mixed with intratubular nephritis. The granular contracted kidney may anatomically be regarded as one morbid condition; clinically, the cases differ greatly, also the antecedents are different.

This disease of kidney is a local expression of several morbid states.

In some cases, watched for years, there has been no cardiac hypertrophy, no vascular disease observed, and in some the arterial tension is low.

It is not yet shown to what extent this renal disease precedes the cardio-vascular; nor the cardiac, the renal. It is certain that the heart may be hypertrophied and vessels much diseased (arterio-capillary-fibrosis), whilst the kidneys are not noticeably, or but little contracted. It is equally certain that the kidneys may be greatly contracted and the heart not hypertrophied.

In a large number of cases there is an anatomical proportion between the two morbid conditions—viz. extreme cardiac hypertrophy and extreme renal contraction; but this is the final issue, not only of the renal and the cardiac disease, but of other concomitant local contractions and thickenings, more or less disseminated throughout the body. To estimate the causes of the heart hypertrophy, we must regard the clinical events throughout, and not those only which are final.

A more or less widespread systemic degeneration of tissues occurs in the morbid state of which granular contracted kidney is, or may be, a component, and this often before albuminuria occurs.

This is shown in many ways and degrees, by breathlessness, prolonged expiration, attributable to lessened pulmonary elasticity (emphysema), disturbed digestion, thinning of the voluntary muscles, looseness, and increase of fat in abdominal walls, bowels less regular (constipation), occasional or more constant occurrence of diuresis, urine variable in colour—from pale and clear without after-deposit to good amber colour with deposit of urates, these varying in amount. At this stage the symptoms are often referred vaguely to latent or so-called suppressed gout; the functions of the lungs and kidneys in varying degrees failing together.

The connective substance (protoplasma) which forms capillary walls, adventitia, and intima of arterioles, is thickened, it becomes more compact, more fibrillated, more rigid;

and this occurs in many organs of the body; in kidneys, lungs, skin, spleen, stomach, heart, retina, spinal cord, brain. The interstitial circulation is hindered by this widespread alteration in the plasma; and consequently nutrition also.

Anæmia and pigmentation of the skin supervene, denoting diminished oxidation and lower vitality of the protoplasm. The sense activities of skin, eye, &c., fail concomitantly with the respiration and muscular energy; the cerebro-spinal functions become disordered, as shown by wandering pains, cramps, or other spasms of muscles, irritability of mind, restlessness, epilepsy, or delirium, &c.; and vaso-motor function fails in many parts from the occurring and various inflammations.

With the spread of the fibrosis there is increasing hindrance of the interstitial circulation, and increased general arterial tension; whilst in the kidney the intertubular fibrosis additionally increases the tension in arterioles and glomeruli. Is it not a correct inference that the heart hypertrophy is much due to the widespread hindering of the circulation through capillaries and capillary walls, &c.? The hypertrophy occurs with this hindrance not only where there is much, but where there is comparatively little *renal* contraction.

The causes of heart hypertrophy must be looked for far and wide. It cannot be forgotten that as elasticity of lung diminishes, the aspiratory power of chest lessens, and with the failing venous circulation, the circulation in the coronary veins is impeded, and nutrition of heart's wall perverted; fat accumulates on right ventricle; there is thickening of interstitial tissue of heart, and fibrosis supervenes in it. Thus, whilst heart muscle is hypertrophied in parts, it may be wasted in the others, until by the increasing obstruction in general circulation, and by the exhaustion of its nutritive power, it dilates.

This course of events doos not exclude many intercurrent conditions, but even probably favours them as concurrently leading to fibroid and other changes in the heart; but we must add that the altered rhythm and mode of impulse of the heart in arterio-capillary-fibrosis are peculiar, and still call for further investigation. It remains to be determined whether there are not in this state conditions in the heart itself leading to enlargement.

CHRONIC NEPHRITIS;

VIZ., THE RELATION BETWEEN THE CHANGES OF CONNECTIVE TISSUE, PARENCHYMA, BLOOD-VESSELS, AND HEART IN THIS DISEASE.[1]

The subject of the following remarks has been variously designated, according to the general aspect which the kidney presented on the post-mortem table, Bright's contracted kidney in the third stage—the granular kidney—the cirrhotic kidney—the kidney of interstitial nephritis. Objections might be made to each of these terms. First as to the use of the terms "Bright's kidney." Bright's investigations had no relation to one particular form of disease of the kidney.

His observations were from a clinical standpoint, and included all cases in which the urine was albuminous during life. The various forms which the kidney presented after death were no further classified by him than as "large and smooth," and "small and granular;" and the intermediate condition, where the kidney was either of normal size and weight, or rather larger or rather smaller than normal, the surface being irregularly smooth or irregularly granular. This intermediate stage of size and surface was vaguely regarded as having relations on the one hand to the large white kidney from which in the process of time it had contracted, and on the other to the small and granular form to which it was supposed to be tending, and to which it would have reached if life had lasted. In this sense therefore the "Bright's kidney" included every form of renal change, the whole series being characterised by albuminous urine during life. Again, the word "contracted" in the term "Bright's contracted kidney" implied, though it did not assert, a community of renal changes, first of swelling, and then of contraction.

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[1] 'Compte-Rendu du Congrès Périodique International des Sciences Médicales, 8me Session, Copenhague, 1884,' tome 1, 1886, Section de Pathologie Générale et d'Anatomie Pathologique, p. 31.

Erroneous as this theory is, and fully as the error of it has been exposed by all modern writers of authority on renal diseases, it is still very tenacious of life, and maintains itself in a degree against these attacks. The permanence of this narrow position is probably mainly due (1) to the habit of regarding the disease of the kidney as of one form and nature, and further as the source and starting-point of the several lesions associated with it throughout the system; as if renal disease was always one, and had always a renal origin; and as if all the morbid changes associated with it were its effects, and had sprung from failing renal function; (2) to the assumption that albumen in the urine indicates one pathology; and (3) to the occurrence post mortem in the several forms of renal disease, of histological changes which are more or less common to all the forms; and hence an erroneous inference that they are of one kind, and have an identical pathology;—as if one should assert that all scars of the skin, seeing that they have common histological characters, have one pathology.

It would seem not to be sufficiently considered that in the nature of the case the morbid forms of histological expression are limited, whatever may be their pathology; and hence these lines of morbid tissue-change will have a tendency to approach each other as they proceed. For example, interstitial nephritis and its results in fibroid tissue and contraction, may occur in kidneys in which the morbific agencies may entirely differ amongst themselves. In catarrhal nephritis there are various degrees of interstitial nephritis, which may produce granulation of the organ, though such interstitial change and granulation may have a quite different meaning from that which occurs in the fibroid kidney which is the special subject of this communication. And the same might be said of the nephritis of scarlatina, of pregnancy, and others. In fact, to repeat what has already been said, in all cases of renal lesions, there will be an approximation less or more to histological changes common to the whole.

Whilst asserting this, it is not my intention to convey the idea that the morbid anatomy of the kidney is not distinctive of the pathological condition out of which it springs; but that a full criticism of differences requires a survey of morbific cause, clinical history, and associated tissue-changes in other organs, as well as of those which occur locally in the kidney; and that without this more complete survey, renal pathology may be expected to remain defective and unprogressive. The truth of this statement will be more evident, though at the same time the prejudice against accepting it will be stronger, in proportion to the limitation of our views to the final results of disease on the kidneys themselves. For however the lesion may have begun, and from whatever cause it may have sprung, its results are destructive; and in the process of destruction they must approximate towards each other; and therefore contraction, granulation, and atrophy, may occur in any nephritis; and the exclusively morbid anatomist will readily find on the post-mortem table, a strong confirmation that there is but one nephritis, one Bright's disease: whilst, in contrast with this barren conclusion, the pathologist, surveying the life-history of these cases, will probably with more approximation to truth find that the word "Bright's disease" has no critical significance, and no value except for the satisfaction of slip-shod and unprogressive therapeutics. It will seem to him probable that the kidney is not necessarily the centre from which all renal pathology can be studied. Without in the least minimising the importance of renal lesions in themselves, and their reactive effects on the organism, we believe it will become more and more evident that antecedent and coincident systemic changes must be more and more studied, before the pathology of nephritis is concluded. Any other exclusive line of inquiry lays us open to the objection that we are seeking "the living among the dead," and exposes us to Goethe's satire—

> "Wer will was Lebendig's erkennen und beschreiben,
> Sucht erst den Geist heraus zu treiben,
> Dann hat er die Theile in seiner Hand
> Fehlt leider! nur das geistige Band."

With these preliminary remarks, which apply, *mutatis mutandis*, to all pathological terms which would convey the idea that destructive changes in the kidney have all a local origin, and constitute the chief pathological entity, I pass on to that which, as I have said, is the special subject of my communication, namely, Fibrosis, or Arterio-capillary-fibrosis of the kidney, as it occurs at, or after, the middle period of life.

And here I leave on one side those forms of nephritis which are variously termed parenchymatous nephritis, albuminous nephritis, tubular nephritis, amyloid change, surgical kidney, scrofulous kidney, nephritis of pregnancy, and such other forms as confessedly have a more limited and local pathology.

In the year 1872, my friend Dr. Sutton and myself,—having clinically observed that cardiac hypertrophy of the left ventricle, without valvular disease, and of the same character as that which goes with the contracted kidney of Bright's disease, might occur without renal change, or might precede it,—set ourselves to inquire into the pathology of these cases, which had hitherto been unclassified. Up to that time, cardiac hypertrophy with renal fibrosis was explained on the theory that the blood, being imperfectly depurated by the kidney, caused a spasm, and subsequent hypertrophy of vessels, which prevented the flow of impure blood through the organs, and called for increased power on the part of the heart, to meet this difficulty.

The insufficiency of this theory became at once apparent, when it was seen that the same cardiac hypertrophy might precede any sign of renal change, and might occur before there was any evidence of defective renal excretion; and to be sure of this, we were not satisfied with the ordinary examination of the urine, but we obtained through a high authority[1] a complete analysis of such urine for twenty-four hours, which showed that there was no noticeable defect in composition. This, therefore, as it may be called "glaring instance" of the fallacy of the old theory, suggested another source of cardiac hypertrophy, outside the kidney; and this was found in the condition of the systemic arterioles and capillaries. These were found in such cases to have undergone various changes. The intima was thickened, the adventitia thickened, and often not to be distinguished from the surrounding connective tissue, the smaller vessels being matted into the connective tissue by a fibroid felt-like hyaline substance; the muscular coat was also variously altered. Even where apparently normal, the nuclei of the muscle-cells did not absorb coloring matter so readily as in healthy vessels. This layer often seemed relatively increased, and might in some cases have been actually hypertrophied.

But more often, even with the increased thickness, there was a morbid change in the muscle-cells; their nuclei were becoming spindle-shaped, or atrophied, or reduced to small globular bodies, having a high refraction like fat.

We summarised our investigations as follows: (1) Kidneys often much contracted, heart much hypertrophied, minute arteries and capillaries proportionately thickened by "hyaline-fibroid" formation. (2) Kidneys little contracted, but heart much hypertrophied, minute arteries and capillaries much thickened by "hyaline-fibroid" substance. (3) Kidneys healthy, whilst heart much hypertrophied, and minute arteries and capillaries much thickened by "hyaline-fibroid" substance.

It is admitted that about the middle period of life, vascular changes in the brain may be fatal from hæmorrhage and the like, with extreme hypertrophy of the left ventricle of the heart; and on the post-mortem table, the kidneys may show but little change; often no more than early granulation with adherent capsule. Yet hitherto no one has referred the cardiac hypertrophy in these cases to defective renal function. In fact, generally the post-mortem record has run thus: "Left ventricle much thickened; kidneys but little affected."

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[1] Dr. Stevenson.

Two fallacies have hitherto beset an open inquiry into this form of renal pathology. The one, that where the renal changes are marked, all the attendant systemic changes have been, without reserve or limit, vaguely referred to the kidney as their source. The other, that though in a given case the systemic changes may have been of a similar character to those in the former case, yet, if the kidney changes were not marked, no relation was suspected between the two sets of conditions. The assumption that the fibroid change in the kidney, of the form of which I am now speaking, has a local origin, appears to us to have prevented an impartial study of all the circumstances of its origin, and course, and complications. It is still widely assumed that this renal change has a more or less acute beginning in inflammation.

Now, although obviously the kidney at any period of life, and more commonly if the health be weakened, may become the seat of inflammation, the fibroid change in question is not inflammatory. There is no acute stage, no acute hyperæmia, there is no diapedesis of leucocytes and blood-cells characterising ordinary inflammation; no local or general symptoms indicating nephritis, as a necssary part of the process.

The first abnormal departure from health as regards the kidney being no more than a diuresis, the urine at that time presenting no morbid characters, except a somewhat lower sp. gr., there are no deposits of any inflammatory echdysis, no caots, no leucocytes, no epithelium. But coincidently with this early stage, and often even preceding it, there may be for months or years marked systemic changes throughout the body; loss of weight; loss of colour, the complexion becoming greyish; symptoms of failing nutrition in different organs, varying in different individuals; skin less elastic; changes in brain-power or spinal power; dyspepsia; shortness of breath; signs of cardiac hypertropy.

This train of events may go on in a vague way from month to month, without any recognised pathological basis, until albumen is found in the urine; and then, according to current view, the case is called "Bright's disease," and no further investigation seems required. Yet at this very stage where all investigation has hitherto stopped, we believe that

Lucina should rather have been invoked than Atropos, and that, instead of cutting short the inquiry, a more lucid pathology would have combined the whole facts into one state, affecting the organism throughout, at least in the vascular area, and that instead of combining the thickened heart and the contracted kidney together directly, the whole man should be placed between the two, and is their proper nexus. That this renal change is not inflammatory, is generally admitted by English pathologists. Dr. Greenfield, Professor of Pathology at Edinburgh, thinks it an atrophic process.[1] "The primary or earliest change," he says "is a fibroid change in the arterial walls, which especially affects the afferent arterioles" (of the Malpighian tufts). "Further," he adds, "if we carefully inquire into the history of the renal changes, we find that at least two forms of disease are grouped under this common name of Renal Cirrhosis, the one essentially of a chronic inflammatory nature, the other due to an atrophic process, dependent on a primary arterial degeneration. It is true that we find these two forms intermingled in some cases, but in others they appear absolutely distinct. In the chronic atrophic form, the primary change is a chronic peri- and endo-arterial fibrous thickening. Strictly speaking, this is not an interstitial nephritis at all, though some intercurrent inflammation may occur."

Although these quotations set forth the most recent views of one of our best pathologists, in respect of the local changes in the fibroid kidney, I have no authority for adducing them as evidence on his part of their relation to similar systemic vascular changes in other organs; although, from our point of view, they have the most direct relation.

Another of our recent writers, and one who contends for the unity of renal diseases, seems to imply from his writing that this fibroid change is the result of nuclear proliferation; a process which extends through the whole structures of the kidney, and into the tubules; an "inflammatory process, of prolonged duration, but of minimum intensity." Describing the changes in the vessels, he writes: "The internal elastic lamina is swollen, its layers are separated, and interspersed with nuclei; within is the much thickened endothelial layer converted into a delicate fibrous tissue; outside the elastic lamina is the muscularis, showing widely separated spindle-shaped nuclei, and looking as if it were œdematous. Outside this again is a cellular connective tissue, in which no adventitial coat can be distinguished from the surrounding connective tissue." Speaking of the connective tissue, he says: "The changes in the capsule of the Malpighian bodies, the adventitial tunics of the vessels, and the basement membranes of the tubes, consist mainly in swelling and hyaline transformation."

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[1] 'Discussion on Albuminuria,' Glasgow Pathological and Clinical Society, p. 71.

He regards the process as of an essentially chronic nature, the changes which take place "partaking more of the character of growth than inflammation."

It may be of interest to remark that the descriptions here given by independent observers of the changes of the renal textures in renal fibrosis, are precisely of the same character, and are expressed in almost identical words, with those employed by ourselves in describing associated changes in other organs.

In describing, for instance, a section of the cord from the lumbar region of a man aged forty-seven, with granular contracted kidney and cardiac hypertrophy, the terms used are "Vessels thickened by hyaline layer;" "Thickened connective tissue, nuclei multiplied;" "Arterioles much thickened, nuclei of their intima multiplied" (nuclear proliferation); "Kidney slightly granular, lungs emphysematous, heart healthy, vessels thickened by fibroid material." Indeed, throughout the paper in which we have described fibroid changes in the vessels of the cord and their surroundings, we have been able to demonstrate vascular changes of precisely the same character as those that occur in the fibroid kidney.

In the spinal cord, as in the kidney, the fibroid change is most marked where the connective tissue is most abundant; extending in cord and in kidney from the adventitia of the arterioles and capillaries into the surrounding connective tissue.

The fibroid material in the cord, as in the kidney, contracts and compresses surrounding tubules, atrophying or destroying them; but leaving many adjacent tubules comparatively normal. In the cord, as in the kidney, it would seem that acute change commonly supervenes on the chronic.

Seeing that so many tubules remain comparatively normal, we are enabled to understand how it is that both cord and kidney may retain much of their functional activity even when they are the seat of very extensive fibroid change. Of the five cases which formed the subject of this communication respecting arterio-capillary fibroid changes in the spinal cord, in two the kidneys were granular and contracted, left ventricle of the heart hypertrophied, no valvular disease; in one, kidney slightly granular, heart not hypertrophied; in two, kidneys not contracted nor granular; in one of these the lungs were the seat of extensive fibroid induration; in the other, brain atrophied, great cardiac hypertrophy.

These particulars show that fibrosis in the cord may occur coincidently with fibrosis of the kidney, or may be in advance of the fibroid change in the kidney, or may occur independently of renal disease.

It may assist us towards a better criticism of our present position, if we for a moment recall the steps which have led to it.

In Bright's time, the morbid changes in the kidney, whatever their nature, so long as they were characterised by the presence of albumen during life, occupied the whole field of clinical thought. The hypertrophied heart and thickened blood-vessels were supposed to be conservative; the one for forcing the impure blood through the tissues, and the other for preventing it. The mere statement now looks almost absurd. It soon became obvious that in a very large class of renal diseases, there are no such changes in the heart and blood-vessels; but, on the contrary, death occurs from an enfeebled circulation, anæmic œdematous textures, uræmia, and exhaustion. And, further, that in what was called by the older physicians the climacteric period of life, from the sixth to the ninth septennial,—the heart, the brain, the cord, the lungs, the skin, the kidneys, and other organs, become liable to degenerative changes in the lines of their arterioles and capillaries, and in the interstitial circulation of the plasma from them, without acute beginnings in any organ, but often with acute intercurrent inflammation.

The heart, despite its liability to these degenerative changes, still exhibits a residual force of nutrition, in the multiplication of its contractile elements (hypertrophy), as might have been anticipated from its earliest relation to the organism.

These degenerative changes, falling upon the vascular area in all parts, would, no doubt, if life could be continued, extinguish the heart itself; but in the earlier stages there is hypertrophy to meet the systemic difficulty; not, we believe, to meet the defect of the renal function pure and simple,—to push on, as it is said, the impure blood,—but to meet the loss of arterio-capillary elasticity, and the hindrance to the flow of plasma through the tissues.

Whilst we can no longer, in one large class of cases, refer the arterio-capillary changes in the various organs, including the kidney itself, to the kidney as the primary seat of disease, and to the consequent uræmia, it is still a question how far a local fibroid change beginning in the kidney, and having its origin there, may lead to systemic arterio-capillary changes of the same character as those which come on idiopathically in later life.

For the present I am content to leave this matter for future inquiry.

It is admitted that granular contracted kidney is rare in the early periods of life. It is also admitted that the kidney may be extremely atrophied by fibroid changes in it of a local kind, in young subjects, and be fatal by unexpected uræmia without cardio-vascular change.

PARTHENOGENETIC TUMOUR

ATTACHED TO THE

MUSCULAR TISSUE OF THE LEFT VENTRICLE OF THE HEART OF A SHEEP.[1]

At the beginning of last year, one of my poor patients, an observant and intelligent man, employed as a slaughterman to one of the great butchers in Newgate Market, brought me the heart and lungs of a sheep, with the apology that although he had killed sheep in great numbers every week for many years, he had never met with the like before. On examining his present I found the peculiarity he had noted depended upon a tumour enclosed in the pericardium, and attached to the left ventricle of the heart. The sheep was a Southdown ewe, five years old, the mother of two lambs, and in good condition, weighing 67 lbs. The pericardium and heart were thickly covered with fat. The tumour weighed rather more than nine ounces. It was attached to the muscular structure of the left ventricle posteriorly. Its surface was irregular, and covered by a layer of the close pericardium, which it had carried before it in its growth. It consisted externally of irregular plates of bone between layers of fibrous membrane. These projected irregularly into its interior, giving it a multilocular character. The large irregular cells thus produced contained recently coagulated blood of a dark colour; some of the cells were entirely filled with such coagula, whilst in others a soft brain-like substance intervened between the coagula and fibrous membrane. A few cells were filled with the soft brain-like substance only. By long maceration of a portion of the tumour in water, several irregular plates of bone were obtained, but no form approximating to any bone of the skeleton could be detected. In many parts they were irregularly cancellated, and contained, as in healthy bone, fat cells with oily contents. The lacunæ and canaliculi of the bone were generally perfectly normal, and often arranged into well-formed Haversian systems. In some parts the lacunæ were larger and more irregular than in healthy bone, and the canaliculi wider, shorter, and more irregularly disposed. The fibrous tissue itself was like that of the dura mater, except that it was less condensed and more easily separable into laminæ. The pulpy brain-like substance was delicately fibrous and adherent to the membrane of the cavities. It consisted of oval nuclei, having a sharp outline, and of a finely fibrous stroma. The transverse diameter of the nuclei was $\frac{1}{4500}$th of an inch, the long diameter $\frac{1}{2500}$th. The fibres were generally uniform, $\frac{1}{7000}$th of an inch in diameter, variously branched; many of them had nuclei attached to one side, rarely embedded in them. The blood-corpuscles of the coagula were normal.

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[1] Reprinted from the 'Guy's Hospital Reports,' 1853, p. 145.

The variety and character of the tissues found in this tumour, and the presence of well-formed bone, separate it from ordinary morbid productions, and impress upon it a physiological rather than a pathological character. Similar tumours occurring in other parts of the human body have been recorded, and their origin has been explained by supposing that one germ has been included within another during the early period of development, whereby one is deprived for a time of the conditions necessary for its growth, but subsequently asserts its independence by a kind of parasitic life. This, though a probable explanation of some of the cases, is not perhaps the only history of these remarkable formations. The late increase of knowledge respecting these subjects, would lead us to believe that a portion of the original germ mass of the impregnated ovum may fail to be included in the formation of a tissue, and thus give rise to an isolated centre of nutrition, which, from the plastic force it inherits, would imperfectly exhibit the general processes of development. A tumour so produced, however imperfect, would be similar in its origin to the individuals of a species multiplied by parthenogenesis, as described by Professor Owen, and hence I have applied the term "parthenogenetic" to the tumour in question.

Such productions are very rare, but their origin seems to elucidate the history of other and more simple forms.

A CASE

OF

INTERMITTENT HÆMATINURIA.[1]

WITH REMARKS.

THIS disease is characterised by the passing, at intervals, of urine of a dark colour, looking as if it contained blood; no blood-corpuscles, however, are present. The urine is, in fact, of a red Burgundy-wine colour, and is often turbid, forming a dark-coloured sediment on cooling. It coagulates by heat and nitric acid. The specific gravity is generally higher than that of normal urine. On examining it under the microscope we notice granules scattered or aggregated into masses, granular casts of the tubules, a few degenerated epithelium-cells, and crystals of oxalate of lime. No blood-corpuscles, or, if any, merely a stray or a doubtful one, are to be seen. The granules, when carefully examined, are found to consist chiefly of very small prismatic crystals of hæmatin; and even such granules as are not so distinctly crystalline are, on changing the focus, seen to have a somewhat similar crystalline appearance. When the crystals and granular matter are thrown slightly out of focus their colour changes from the claret hue of hæmatin to a pale green.

One of the most striking points in the clinical history of this affection is its intermittent character, the urine suddenly, and within an hour or two, changing from a deep blood colour to a pale straw colour.

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[1] Reprinted from the 'Guy's Hospital Reports,' 1866, p. 381.

The following case afforded many opportunities of observing these changes. It was usually the early morning urine which was abnormal. The urine after one or two o'clock in the afternoon usually presented the normal characters. The intermittent character of the affection and the aspect of the urine have led Dr. Harley to name this disease intermittent hæmaturia. I prefer, however, to call it intermittent hæmatinuria. The following case differs from those described by Vögel, in which the urine contained hæmato-globuline, in that the patient was not suffering from any exhaustive fever, and the hæmatin was visible in the urine in a crystalline form.

For the following particulars of this case I an indebted to my clinical clerks, Mr. Algernon Ewen and Mr. Henry Denne.

James G—, æt. 33, a labourer, was admitted January 31st, 1866, into the Clinical Ward, Guy's Hospital, under the care of Dr. Gull. He is a married man. He states that he has always enjoyed tolerably good health. His habits have been regular. He is an anæmic-looking man. The history of his present ailment is as follows:—Five months ago he noticed, when passing his water, that it very much resembled blood in colour. At that time he was employed in leather-dyeing, and in consequence he was very much exposed to "wet"—constantly more or less wet through "the water being frequently sluiced over me." He continued to pass the bloody-looking urine for two days, after which his urine assumed its normal appearance. He did not discontinue his work while the urine presented this peculiar character. Three weeks before admission he again observed a similar condition of his urine. This time he was compelled to leave off work, on account of a severe pain in his loins, which prevented him standing. The supposed bloody condition of his urine has continued ever since. If he exerts himself, as in lifting a weight or walking much, he is sure to find his urine more highly coloured, and more like pure blood.

On admission. He looks anæmic, but is apparently a well-nourished man, but he thinks he has wasted lately. His skin is moist. Temperature 97·5°. Some of the cervical glands on the left side are enlarged and hard. His tongue is moist and clean. Pulse 60, sharp and small. Respirations 24. Heart perfectly natural, as regards rhythm and impulse and sounds. Lungs apparently healthy. He complains of a shooting pain, which extends from the umbilicus into the loins, and is increased by coughing or drawing a deep breath. There is no pain in the course of the ureters, and there is no vomiting. The bowels have not been opened for two days. There is no œdema about the legs. The urine is of a dark mahogany colour, slightly acid, of sp. gr. 1014. On standing it deposits a light cloudy sediment.

There is no change on the addition of ammonia. When treated with sulphate of copper and liquor potassæ it throws down a blue precipitate tinged with black. A heavy gelatinous-looking precipitate is obtained on boiling, which is not dissolved, but rather increased in quantity, by the addition of nitric acid. There is no evidence of bile, by either the nitric acid test or that with sulphuric acid and sugar. Under the microscope a very few granular epithelial cells and some granular urates are seen, but no blood-corpuscles.

February 2nd.—The urine has a sp. gr. of 1024, is of an amber colour, and deposits a thick, whitish, flocculent precipitate on cooling. On boiling it a gelatinous precipitate is formed, which disappears on the addition of nitric acid.

Sumat Acidi Nitrici diluti, ♏xv,
Infusi Cascarillæ, ℥iss, bis die;

Pil. Rhei comp. gr. v, omni nocte.

3rd.—He got up this morning. After walking about the ward for a time he was seized with rigors, and with pains in his loins extending into his abdomen. He passed some very dark-coloured urine—about three quarters of an ounce. This is opaque, almost of an indigo colour, and clouded with albumen. There is not a quantity of it sufficient to enable the specific gravity to be taken. The microscope shows granular casts, urates, and granular-looking cells, but no blood-corpuscles.

4th.—Urine highly albuminous, sp. gr. 1033.

5th.—He has passed 34 oz. of urine in twenty-four hours; that passed during the night is of a mahogany colour, sp. gr. 1024; that which was passed this morning is of a normal amber colour, sp. gr. 1010.

On examining the dark-coloured portion under the microscope we observe no blood-corpuscles and no casts, but numerous crystals of hæmatin, having a brilliant red colour, a few crystals of oxalate of lime, and much granular matter. A gelatinous precipitate appears on boiling, and becomes much denser on the addition of nitric acid. The amber-coloured urine also gives a gelatinous precipitate by heat, which disappears entirely on the addition of nitric acid.

6th.—He has passed, during the night, about 30 oz. of amber-coloured urine, sp. gr. 1015. With heat and nitric acid it yields the same reaction as the amber-coloured urine of yesterday. This morning he has passed about half an ounce of bloody-looking urine, sp. gr. 1030. The microscope shows no blood-corpuscles, but crystals of oxalate of lime, granular epithelial cells, and several crystals of hæmatin.

7th.—He is again passing amber-coloured urine, slightly acid, and of sp. gr. 1016. With heat and nitric acid there is the same reaction as before, that is, the urine contains no albumen.

8th.—Since yesterday he has passed about 13 oz. of dark indigo-coloured urine, besides 20 oz. of amber-coloured urine. The urine is of sp. gr. 1030, and loaded with albumen. It presents no blood-corpuscles, but plenty of hæmatin crystals.

10th.—This morning he has passed dark mahogany-coloured urine. This measures 10 oz., its sp. gr. is 1025. It deposits a thick brownish sediment on standing. The microscope shows hæmatin crystals, oxalate of lime crystals, and distinct casts, the matter forming which is of a dark colour.

13th.—The urine is of a dark mahogany colour, highly albuminous; quantity in twelve hours 16 oz., sp. gr. 1028.

14th.—Early in the morning he passed 16 oz. of amber-coloured urine, sp. gr. 1014, containing no albumen. Since he has been up this morning he has passed 2½ oz. of dark indigo-like urine, loaded with albumen, sp. gr. 1030. This contains no blood-corpuscles, but hæmatin crystals and oxalates in abundance.

15th.—In the night he passed 21 oz. of straw-coloured urine, of sp. gr. 1012. To-day he passed 18 oz. of dark mahogany-coloured urine, of sp. gr. 1017. Each kind of urine possesses the same characters as before.

16th.—This morning he passed 14 oz. of amber-coloured urine, of sp. gr. 1025. The reaction with heat and nitric acid is the same as before.

18th.—From 5 p.m. until 9 p.m. he passed about 14 oz. of dark mahogany-coloured urine, of sp. gr. 1020, containing no blood-corpuscles.

19th.—During the night he passed 20 oz. of amber-coloured urine, of sp. gr. 1010. He has been out of bed this morning. Up to 2 p.m. he has voided 18 oz. of dark indigo-like urine, of sp. gr. 1023. The microscope shows no blood-corpuscles, but numberless crystals of oxalate of lime, and uric acid crystals, as well as granular casts and crystals of hæmatin.

20th.—The urine passed in the night is amber coloured; that voided this morning is mahogany coloured.

21st.—He has not passed any mahogany-coloured urine to-day, the secretion being of a dark amber colour; it is not coagulated, but it is cleared by heat, and no precipitate is thrown down on adding nitric acid. On standing it deposits an abundance of pink lithates.

22nd.—His darkest urine is now of an amber colour, and but very little darker than the urine that he ordinarily passes. Its sp. gr. is 1026. No change is produced by heat or by nitric acid. Boiled with liquor potassæ, it does not become darker. On standing it gives no appreciable deposit.

23rd.—Yesterday, before going to bed, he passed 14 oz. of clear, amber-coloured urine, of sp. gr. 1014. In the night he passed 18 oz. of light amber-coloured urine, sp. gr. 1005. This morning he passed 15 oz. of urine of a clear amber colour, and of sp. gr. 1020. On standing this gives no deposit.

24th.—His darkest urine is now of a clear amber colour, and of sp. gr. 1020.

25th.—No dark urine is now passed.

26th.—The morning urine is of an amber colour, and of sp. gr. 1020. It has an acid reaction. It is not changed by applying heat or nitric acid. On standing twenty-four hours it throws down a flocculent deposit, consisting of oxalate of lime crystals, granular-looking cells, and pavement epithelial cells. The lymphatic glands below and behind the left angle of the inferior maxilla are enlarged and tender, and there is a blush on the skin over them.

27th.—The urine passed this morning is of a clear amber colour, and of sp. gr. 1022. There is none which is darker.

28th.—On getting up this morning he felt faint and trembling, and passed some urine, of sp. gr. 1020, not much darker than usual, but rather smoky. It is rendered faintly opaque by heat, and is not cleared by nitric acid. On standing it deposits numerous crystals of oxalate of lime, seen by the microscope.

March 1st.—The glandular swelling has not diminished in size. He has had some shivering this morning and pain in the loins, lasting an hour or two. During this time he passed 6½ oz. of dark port-wine-coloured urine. It coagulates on applying heat and on adding nitric acid. Under the microscope no blood-corpuscles are seen, but oxalate of lime, granular cells, and granules.

2nd.—His urine presents three varieties—that passed yesterday is smoky, of sp. gr. 1016, and amber coloured; that passed in the night is of a light mahogany colour, and of sp. gr. 1014; that passed this morning is of a dark mahogany colour, and of sp. gr. 1020. Each variety is coagulated by heat and by nitric acid. There is very little deposit on standing. No blood-corpuscles are seen.

3rd.—His darkest urine to-day is of a smoky amber colour, its sp. gr. is 1016. It is coagulated by heat or nitric acid. It contains crystals of hæmatin, but no blood-corpuscles.

4th.—The urine passed in the night is of a dark amber colour; that passed this morning is of a dark mahogany colour. Ordered Tr. Cinchonæ co. ʒij ex Julep. Menthæ ter die.

5th.—This morning's urine is of a dark mahogany colour, its sp. gr. is 1020. It is coagulated by heat and by nitric acid.

6th.—The darkest urine is of a clear amber colour, sp. gr. 1020. It is unchanged by heat and nitric acid.

7th.—The darkest urine is of an amber colour and turbid, its sp. gr. is 1024. It has an acid reaction. It is cleared by heat, but not cleared by potash, and is not coagulated by nitric acid. The deposit consists of amorphous granules of lithates and oxalate of lime.

8th.—All the specimens of urine are clear, sp. gr. 1012. No change by heat or by nitric acid.

10th.—During the 9th he passed 20 oz. of clear amber-coloured urine, sp. gr. 1016. During the night of the 9th 16 oz. of clear amber-coloured urine, sp. gr. 1018. This morning, 16 oz. of clear amber-coloured urine, sp. gr. 1020. Each specimen is unchanged by heat or by nitric acid.

11th and 12th.—The urine is of a clear amber colour, sp. gr. 1017 and 1022.

13th.—The urine is of a clear amber colour. That passed in the night has a sp. gr. of 1013, that during the day of 1024. There is no change by heat or nitric acid. No trace of sugar is to be detected.

14th.—This morning (the temperature being much lower than it was yesterday) he shivered, felt faint, and trembled when getting up.

Yesterday (on the 13th) from 11 a.m. to 1 p.m., he passed 17 oz. of clear amber-coloured urine, of sp. gr. 1016. Last night, from 8 p.m. to 6 a.m., he voided 26 oz. of urine, of sp. gr. 1012.

This morning, from 6 a.m., he has passed 11 oz. of urine, of a reddish-amber colour, sp. gr. 1028. The last specimen coagulates when heated and when nitric acid is added to it. On standing twenty-four hours, a flocculent precipitate is deposited, which, under the microscope, is seen to consist of oxalate of lime crystals, crystals of hæmatin, squamous epithelium, granules, and granular-looking cells; no casts nor blood-corpuscles are to be discovered.

15th.—Yesterday, 11 a.m. to 8 p.m., he passed 17 oz. of a clear amber-coloured urine, sp. gr. 1015. In the night, 8 p.m. to 6 a.m., 13 oz., of a clear amber colour, sp. gr. 1024. This morning, 6 a.m. to 11 a.m., 7½ oz., of a pale amber colour, sp. gr. 1024. These specimens are unchanged by heat and nitric acid.

16th.—Yesterday, 11 a.m. to 8 p.m., he passed 17 oz., of a clear amber colour, sp. gr. 1019. 8 p.m. to 6 a.m., 24 oz., of a clear amber colour, sp. gr. 1018. 6 a.m. to 11 a.m., 10 oz., of a clear amber colour, sp. gr. 1024. Each of these specimens is clouded by heat, but cleared by nitric acid.

17th.—The urine in all essential particulars the same as yesterday.

18th.—All the urine passed is of a clear amber colour. That voided in the morning is neither lighter nor darker than that passed at other times.

19th.—Yesterday, 11 a.m. to 8 p.m., he passed 18 oz., of an amber colour, not very clear, sp. gr. 1022. 8 p.m. to 6 a.m. he passed 22 oz., clear, of an amber colour. sp. gr. 1020. 6 a.m. to 11 a.m., 13 oz., pale amber colour, sp. gr. 1007. These specimens are all unchanged by heat and nitric acid.

20th.—From 11 a.m. yesterday to 6 a.m. to-day he passed 29 oz., of a clear amber colour, sp. gr. 1022. This morning, 6 a.m. to 11 a.m., 13 oz. pale straw-coloured urine, sp. gr. 1010.

21st.—All his urine is clear and of a normal colour.

22nd.—The urine presents exactly the same characters.

23rd.—Yesterday, 11 a.m. to 8 p.m., he passed 1 pint, of a pale amber colour, not very clear, sp. gr. 1020. 8 p.m. to 6 a.m., 1 pint, of a straw colour, not clear, sp. gr. 1015. This morning, 6 a.m. to 11 a.m., 8 oz., of a straw colour, clear, sp. gr. 1014. Each of these specimens has a faintly acid reaction, and is rendered cloudy by heat, but cleared by nitric acid.

24th.—Urine all normal in colour.

25th.—Urine all normal in colour.

26th.—Urine normal.

He leaves the hospital, being convalescent.

In this case we had a good opportunity of observing the intermittent character of the disease. The attacks generally took the following course. During two or more days the patient voided urine containing hæmatin; then, suddenly, the urine changed, and assumed its normal characters. At another time he passed this dark urine for one day only, the urine being normal on the following day.

These changes in the urine were, however, still more striking and sudden than would appear from the above description; for while the urine that was passed in the early morning was found to contain hæmatin, that which was passed at noon or during the latter part of the day was healthy, and so it continued for as many as eight days. Such is the usual clinical history of these intermittent attacks.

In the first case recorded in Dr. Harley's paper it is stated that the patient suddenly passed five or six ounces of urine of a dark red, or chocolate colour. This used to occur once in the twenty-four hours during two or three days, and then as suddenly disappeared. On one occasion this patient passed in the morning normal urine, at two o'clock he voided urine containing hæmatin, and in the evening of the same day he again passed healthy urine.

In the second case recorded by Dr. Harley it is stated that the urine assumed the colour of blood, a symptom which greatly alarmed the patient, especially as it occurred about three times a week during the whole of the winter.

This very dark urine has a high specific gravity. In the above case it was, at one time, as high as 1033. Dr. Harley has found that in these cases the urine contains an excess of urea.

The albumen present appears to be in an altered condition. On boiling the urine it coagulates and becomes opaque. On adding a few drops of nitric acid it becomes still more opaque, but if an excess of nitric acid be added the precipitate is dissolved. It is considered, therefore, that this precipitate is due, not to albumen, but to globulin. Thus, the great characteristic of this dark urine is that it contains hæmatin and globulin in a free state.

An important point for consideration is the cause of this disease. It appears that there is a predisposing and also an exciting cause. In this case the exciting cause was exposure to cold and wet. In the second case given in Dr. Harley's paper exposure to cold seems also to have been the exciting cause of the disease. In Dr. Fuller's case, brought before the Royal Medical and Chirurgical Society by Dr. Dickinson, it is stated that the patient had been exposed to cold and damp. The report further says that if the patient, after getting up apparently well and going to his work, chanced to be exposed to cold, he was attacked with shivering and retching, and that an hour or so afterwards he passed urine black like porter. In other cases there has been a history of ague, and the disease has appeared to be in some way connected with malarial poison. But these are not the only causes. There is reason for thinking that a blow or injury to the loins may give rise to this complaint. Thus, a young lady, in getting into a railway carriage, fell, and hurt her back. Soon afterwards she passed dark bloody-looking urine. I carefully examined the secretion by the aid of the microscope, but found in it no blood-corpuscles, and only the granular pigment-matter of disintegrated blood-corpuscles.

The attacks usually come on with indications of considerable constitutional disturbance. It may be noticed that, in the above report, the patient is stated to have shivered, and to have had pain in his loins so severely that he was compelled to leave off work, and it was soon after this that he voided the bloody-looking urine. Another time his attack came on with trembling and faintness. A third time he shivered, felt pain in his loins, and soon afterwards passed the very dark urine.

Besides the exciting cause there appears to be a predisposing cause in these cases, for we are well aware that many people may be exposed to wet and cold without, as a consequence, suffering from hæmatinuria. We are therefore led to assume that there is a condition of the system which, under certain circumstances, is favorable to the development of this affection; and it will probably hereafter be found that it is in this predisposition that the essence of the disease lies.

The question now arises, what do we know of the pathology of this disease? Some suppose that it is dependent on an altered condition of the blood, and that the primary changes take place in the blood itself. An argument in favour of this view is found in the fact that a somewhat similar condition of the urine is sometimes seen in scurvy, in typhus fever, and also after inhaling arseniuretted hydrogen.

In the present state of our knowledge it is impossible to say that the primary change does not take place in the blood, yet there are positive indications that the kidneys are at fault. The pains in the loins, the condition of the urine, and the casts which are present, all clearly show that the kidneys are affected. In order, however, to point out what appears to me to be the pathology of this disease, I must here briefly allude to the functions of the kidney.

I will not discuss the large question of the physiology of these organs, but will simply confine myself to a few observations on the subject.

The theory of the present day is, that water permeates the walls of the capillaries forming the Malpighian tuft, and runs down the urinary tubule. The epithelial cells which lie in the tubule are supposed to attract urea, uric acid, and other solid constituents of the urine from the blood. These solid constituents are then dissolved in the water coming down from the Malpighian tufts. Now, I do not believe this theory, nor did I when I lectured on physiology some years ago. I consider that the great function of the kidneys resides within the Malpighian bodies, and also that the tubules are infinitely more dynamical than is generally supposed. It appears to me that the tubule is secretive and absorptive, but not excretive. It is thought that the epithelial cells lining the tubules extract the solid constituents of the urine from the blood, and that, while doing so, they are themselves shed from the basement membrane. If, however, we examine healthy-looking urine, we find that it does not contain any evidence of such a process of desquamation, for in normal urine it is very rare indeed to observe the epithelial cells of the kidneys.

As is well known, one of the great functions of the kidneys is to form the colouring matter of the urine from the colouring matter of the blood. How that change is accomplished we do not yet know; but we have good evidence to show that the kidneys must be in a healthy condition in order that the colouring matter of the urine may be formed. This we see illustrated by watching what occurs when the kidneys are affected after scarlet fever. We frequently see hæmatinuria as a sequela of this disease. The usual history is as follows:—After an attack of scarlet fever the child passes albumen and blood in its urine. The microscope shows that blood-corpuscles are present. The affection is a simple hæmorrhage from the kidneys. But when the child advances towards recovery, and the kidneys begin to resume their functions, although albumen may be present in the urine, we no longer find blood-corpuscles, these being replaced by hæmatin. The urine is dusky, but contains no blood-corpuscles. The kidneys have regained their functions so far that they can now break up the blood-corpuscles. In the next stage the urine, still containing albumen in small quantities, presents uric acid and urates, and we then know that the kidneys are beginning to recover themselves. In the fourth stage the urine contains no albumen, but urates, urea, and its natural colouring matter. The kidneys have then totally regained their functions, and we have seen, step by step, the dynamical power of these organs return.

We may illustrate these changes by the following diagram :

Urine after scarlet fever, contains—

1st. Albumen and blood.
2nd. Albumen and hæmatin.
3rd. Albumen and uric acid.
4th. No albumen, but urates, urea, and the colouring matter of the urine.

In hæmatinuria some of the dynamical properties of the kidneys appear to be lost. I say *some,* because the organs still retain sufficient power to cast out an excess of urea, but have lost the power to convert the hæmatin into the normal colouring matter of the urine. They ought to eliminate the hæmatin in the condition of urine pigment ; instead of that, they eliminate the hæmatin itself.

As, however, the kidneys regain their functions they again convert the hæmatin into the ordinary colouring matter of the urine.

Thus, then, it appears to me that, whatever may be the primary change in cases of this disease, we have at least good evidence to show that the kidneys are affected.

This is the local disease, and the one that must attract our attention. For everyday experience shows that a man may have his tissues greatly diseased, and his blood greatly altered (as in gout), and yet that he may be free from symptoms until some vital organ is affected. Moreover, even then the patient is in danger, not because he has diseased blood or diseased tissues, but because an organ essential to life is losing its functions.

SECTION III.

DISEASES OF THE RESPIRATORY SYSTEM.

ON

DESTRUCTIVE CHANGES IN THE LUNG

FROM

DISEASES IN THE MEDIASTINUM

INVADING OR COMPRESSING THE

PNEUMOGASTRIC NERVES AND PULMONARY PLEXUS.[1]

Physiologists have proved by experiments on animals that, after division of *both* pneumogastric nerves, the lungs undergo destructive changes.

The first effect of the operation is a lowering of the frequency of the respirations. After a few hours the pulmonary tissue becomes congested, and then follows effusion of serum into the air-cells and bronchial tubes. At this stage most of the animals experimented upon die, but in such as survive, œdema of the tissue is followed by purulent infiltration (red and grey hepatization) and often by gangrene.

These are the effects of division of *both* pneumogastric nerves. If only *one* is divided, the damage to the lungs is not so certain. Reid, who very fully investigated this subject, reports that he removed a portion of the *par vagum* in fourteen animals, and never observed any morbid structural change that could be attributed to the section of the nerve. None of these animals appeared to suffer any bad consequences. The dogs breathed easily, and ate and digested as before; the rabbits also were as lively and active as ever, after the operation. No morbid changes could be observed in the lungs of the calves and dogs after death. Three rabbits which were allowed to live a fortnight were apparently in perfect health when killed, and no structural difference could be perceived between the lungs of opposite sides. A rabbit which lived three months died of recent pneumonia affecting *both* lungs. From these experiments Reid concludes that the morbid changes desbribed by others must have been accidental. Majendie,[1] it is well known, expressed an opinion contrary to that here quoted from Reid. He says, "Vous savez que dans le cas ou l'on ne coupe qu'un seul nerf on trouve après quelques jours le poumon profondemment altéré et réduit souvent à une masse hépatisée; si l'animal ne meurt pas, c'est que l'action d'un seul poumon est suffisante pour l'entretien de la vie." Again, he says, "Quand on coupe un seul nerf de la huitième paire le poumon auquel appartient ce nerf devient le siège d'altérations de plus en plus graves. Son tissu s'enflamme (c'est l'expression consacrée) et bientôt il devient impropre à la respiration." In one experiment he found the lung on the side of the divided nerve healthy, the animal having survived the operation six months. This contradictory fact he endeavours to explain by hinting that there may have been a reunion and repair of the nerve, or that the damaged tissue may by absorption have regained its normal state. Wundt,[2] who instituted a number of careful experiments upon the influence of division of the pneumogastric nerves upon the respiratory organs, makes no further observations on the effects of division of one nerve than that the number of respirations is lessened and their rhythm irregular. My friend and colleague Dr. Pavy, who in prosecuting his inquiry into the production of diabetes has in numerous instances divided the pneumogastric nerves, informs me that whilst pneumonia follows section of both nerves, injury of one nerve does not produce any obvious effects.[3]

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[1] Reprinted from the 'Guy's Hospital Reports,' 1859, p. 307.

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[1] Majendie, 'Sur les phénomènes vitales,' pp. 203 and 224.

[2] Müller's 'Archiv,' 1855.

[3] The following note has been kindly given me by Dr. Pavy:

"Division of one pneumogastric does not produce any sensible pulmonary disturbance. In one instance an animal was killed three weeks after the

The free communication between the two vagi in the pulmonary plexus is the probable reason of this difference in the results between the division of one and both nerves. By it each nerve can maintain the normal integrity of the plexus, and only when both are injured will morbid effects result.

These experimental deductions are, however, only true as respects injuries of the trunks of the pneumogastrics as they lie in the neck, for it is there only that experiments on these nerves are possible. The results are different when the nerves and their plexus are injured by disease lower down at the root of the lung, because not only are there large branches from the sympathetic at that part, injury of which would have an influence upon the morbid processes, but because the branches going to either lung may be individually implicated, and consequently the corresponding lung be directly deprived of its nervous supply.

The following cases illustrate this.

CASE I.—*Aneurism of the left side of the arch of the aorta pressing upon the left pneumogastric nerve and upper part of pulmonary plexus; sloughing pneumonia of the left lung.*

George R—, æt. 35, admitted into Guy's Hospital, under the care of my colleague Dr. Addison, March 25th, 1850; a sailor, and up to the age of thirty-three always enjoyed good health. At that time he had a sudden attack of paralysis of the right side, with loss of speech. He gradually recovered, but was never again able to read or write, both of which he did tolerably well before the seizure. His face was thin and haggard, expression anxious;

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division of one nerve, and nothing unnatural in the chest was noticed on examination.

"Division of both pneumogastrics occasions great distress of breathing; and pneumonia is invariably found if the animal live long enough, which is almost constantly the case. The inflammation is distributed in patches over the lungs, and in forty-eight hours after the operation I have noticed the lung-tissue broken down into small purulent cavities—the pus-globules having been recognised by the microscope.

"Division of the carotid sympathetic in the rabbit (where it is distinct from the pneumogastric) does not visibly affect the lungs.

"Division of the ascending branches of the superior thoracic ganglion of the sympathetic invariably leads to a fatal pleurisy on the side on which the operation is performed. The pleurisy is limited to the pleura parietalis, the lungs and membrane covering them presenting a perfectly natural appearance."

body emaciated; voice husky; respiration hurried, and impeded by laryngeal obstruction. Breathlessness. He complained that for a month he had had an uneasy tickling sensation in the throat, causing frequent cough. There was no pain on either side of the chest. Some difficulty in deglutition, more for fluids than for solids. Skin hot and sweating. Tongue furred. Pulse 100, small and feeble. The left side of the chest was dull, on percussion, throughout. No tactile vibration. No vesicular murmur. No bronchial sounds, except near the apex, where loud bronchial breathing was audible. Except this, the whole of the left side of the chest seemed impervious to air. The right lung appeared to be abnormally resonant. Respiration puerile. Mucous râles in larger tubes. Sputa rather abundant, muco-purulent. Under the cartilage of the second rib on the left side, a soft double (aneurismal) whiz was heard. The diagnosis was aneurism of the arch of the aorta, phthisical disease of the left lung, and pleurisy. He died exhausted on the 5th of April.

Post-mortem examination.—Right lung healthy. Left lung irregularly consolidated; the tissue grey. In the upper lobe, irregular cavities formed by the breaking up of the tissue, and communicating with the bronchial tubes. The tubes themselves were filled with muco-purulent secretion. Some effusion into the pleura. Heart healthy. At the arch of the aorta, on the left side, there was an aneurism of the size of a large orange, which had extended downwards and backwards, and compressed the left pneumogastric nerve and the adjacent branches of the pulmonary plexus. The posterior part of the sac was formed by the bodies of the first, second, and third dorsal vertebræ. The sac itself had not burst. In the fissure of Sylvius, on the left side, and covering some of the convolutions of the island of Reil, was a tough yellowish substance, the remains of a large clot. The grey matter beneath was partially absorbed, and the yellow substance dipped down into the corpus striatum, in which there was a spot of softening.

In this case, as in the two others I have to relate, the physical diagnosis was obscured by the state of the bronchial tubes. When the nerves of the pulmonary plexus are injured, paralysis of the tubes follows; they gradually become filled with exudation, which they cannot expel, and hence are impervious to air. Besides this, as I shall have again to notice under the third case, the chest is flattened, probably from atelectasis of the pulmonary tissue, which comes on with the paralysis of the tubes. This and the dulness on percussion and absence of respiratory movement may lead to the diagnosis of chronic pleuritic effusion undergoing absorption, when on post-mortem examination it may be found, as in the above and following cases, that there is a very different state of things, namely, obstruction of the paralysed tubes and chronic pneumonia.

CASE 2.—*Cancer of the œsophagus invading the trunk of the right pneumogastric nerve and the branches of the pulmonary plexus behind the right bronchus; enlarged bronchial glands; ulceration of right bronchus; pneumonic consolidation and commencing gangrene of right lung.*

James R—, æt. 45, admitted into Guy's Hospital, under my care, November 21st, 1854. For some weeks he had had difficult deglutition and pain under lower third of sternum, and at the ensiform cartilage. Emaciation. Cough. Peculiarly offensive muco-purulent expectoration, streaked with blood. Dulness, on percussion, from the lower angle of the scapula to the base of the lung on the right side. Absence of respiratory sounds at the seat of dulness, mucous crepitation and bronchophony above. Puerile respiration over left side. He died on the 29th.

Post-mortem examination.—At the commencement of the œsophagus there was epithelial cancer, ulceration extended down as low as the root of the lungs, but neither the lungs themselves nor the pleura were invaded. The areolar tissue around the œsophagus was infiltrated by the cancer, and especially on the right side, about the right bronchus. The right pneumogastric nerve was at this part of its course implicated in the disease, so that it could not be traced to its distribution. The right lung was extensively consolidated; its lower lobe was infiltrated with a greyish or rather greenish sero-purulent fluid, having an offensive odour. The mucous membrane of the bronchi on this side was intensely congested, the tubes obstructed with muco-purulent secretion. The bronchial glands were enlarged by cancerous deposit in them. From one of these, lying on the right bronchus, the cancer had extended into the tissue of the tube.

Left lung healthy. Larynx healthy. Heart and other viscera healthy.[1]

Remarks.—It could not in this case be so unequivocally inferred, as in the preceding, that the destruction of the lung was alone due to disease of the nerves, for the cancerous affection had extended to the bronchial glands and also invaded the tissue of the bronchi. It may therefore be supposed that there was obstruction of the absorbents and of the bronchial veins, which would influence the result. Although in similar cases the possibility of such complications and their operation cannot be overlooked, there still remains the fact that the nerves were in this case invaded and destroyed, accompanied with those changes in the pulmonary tissue which would follow paralysis of the bronchial tubes.[2]

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[1] This case is recorded by Dr. Habershon, 'Observations on Diseases of the Alimentary Canal,' Case xx.

[2] Whilst writing these notes I was informed by my friend Dr. Wilks that

Case 3.—*Fibrous thickening (malignant?) of the tissue in the mediastinum and around the right bronchus (but not narrowing or compressing it); implication of the right pneumogastric nerve and branches of pulmonary plexus; consolidation and purulent infiltration and sloughing of the pulmonary tissue; small bronchial tubes much dilated and full of muco-purulent secretion.*

Joseph J—, æt. 61, admitted into Guy's Hospital, under my care, January 12th, 1859. He was much emaciated, and had a cachectic aspect. The report he gave of himself was that he had been ill for four months, and that his symptoms began with sharp pain in the right side, without cough. On examining the chest, the right side was found entirely dull on percussion, but the natural resonance of the sternum remained. The whole side was flattened, and the infra-clavicular space depressed. On auscultation, no respiratory murmur nor any bronchial sounds were audible at any part. There was no vocal resonance nor tactile vibration. He had never spat blood. The left side was resonant on percussion throughout. Bronchial râles in the larger tubes. Expectoration muco-purulent, without odour. The diagnosis was chronic pleurisy, with effusion undergoing absorption. Malignant disease was suspected, but no enlarged glands could be found in the axilla or neck, nor were there any symptoms of pressure on the parts in the mediastinum, causing difficult respiration or deglutition. He died on the 19th.

Post-mortem examination.—Old and universal adhesions of right pleura. Fibrous thickening and induration (malignant?) around the bronchus, not narrowing or compressing it, but implicating the trunk of the right pneumogastric nerve and the branches of the pulmonary plexus. The trunk of the nerve was so entirely confounded with the new tissue that it could not be traced through it. The smaller divisions of the bronchial tubes were universally dilated up to the periphery of the lung. They, as well as the larger tubes, were choked with muco-purulent secretion. The pulmonary tissue was consolidated into the state of grey and iron-grey hepatisation. In the lower part of the upper lobe was a large sloughing cavity, from the breaking up of the indurated tissue. The pleura pulmonalis was thickened to the extent of an eighth of an inch in parts. The left lung was healthy. The bronchi on this side were free. Heart and abdominal viscera healthy.

Remarks.—This case affords an excellent illustration of the effects which are referable to paralysis of the pulmonary plexus on one side—accumulation of muco-purulent secretion in the paralysed bronchi, subsequent dilatation of the tubes at their peripheral distribution, concomitant exudation into the air-cells (hepatization), and at length disintegration of

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Dr. Budd had lately read a paper before the Royal Medical and Chirurgical Society, on "Disease of the Lung from Malignant Disease in the Mediastinum." I have not seen any notice of this.

the tissue. It is also worthy of notice that the whole volume of the lung was diminished, as shown by the flattening of the chest and falling in of the infra-clavicular space. This might be partly referable to the contraction of pleuritic adhesions, but it is probable that it was chiefly caused by that atelectasis of the tissue which followed upon paralysis of the bronchi. The steps of this process have been made out on the lungs of animals after division of both pneumogastrics. It seems that the capillaries of the lung, becoming congested, encroach upon the space of the air-cells, and produce an airless and dense state of the pulmonary tissue, with reduction of volume.

The effects of division of the pneumogastric nerves have been referred by physiologists chiefly to paralysis of the bronchi, and not to the destruction of any mysterious influence which the nerves may be supposed to have in the normal state on the pulmonary tissue. Paralysis of the bronchi would include not only the loss of muscular and sensitive function as regards the tubes themselves, but also the loss of the excitor influence of the bronchial surface upon the medulla oblongata. It is to this latter cause especially that Reid attributes the morbid results. He says, "If the congested state of the blood-vessels precede, as we believe, the effusion of the frothy serum, we have next to inquire what is the cause of the retardation of the blood and congestion of the blood-vessels in the lungs. This we were formerly inclined to believe might depend upon paralysis of the muscular fibres of the bronchial tubes, but being unable to obtain any satisfactory evidence of this, we again watched the phenomena more narrowly, and now believe that all the morbid changes observed in the lungs can be traced to diminished frequency of the respiratory muscular movements."[1] This theory, though apparently admissible under the conditions of division of both nerves, is plainly inadequate when the injury is limited to one. It cannot then be diminished frequency of respiration to which the effects are due, for if so, both lungs would suffer equally. And the same may be said of the theory of Fowelin, who refers the morbid changes to gradual paralysis of the heart. We must obviously look to more limited causes when, as in the cases above given, the effects are so limited. There seem to be but two ways in which the morbid changes can occur when one lung only is affected. It cannot be, as we have just said, through alteration in the respiratory rhythm, nor in any alteration in the force or frequency of the heart's action, since these causes would influence both lungs equally. It must, therefore, be either through some direct nutritive disturbance in the pulmonary tissue, as assumed by Schiff, or through paralysis of the bronchi and vessels. The former is an hypothesis which has often been put forth, but is still unsupported by proof, for we know of no way in which the nerves can influence the nutrition of a part but through some alteration of the force of the contractile elements in it or in its vessels. "In the present state of physiology," says Wundt,[1] "it is not possible to see more in the disturbances which follow section of the vagus than a sensitive or motor paralysis, since such are the only modes of action we know of in peripheral nerves." Paralysis of the bronchi, and of the vessels, is admitted to be a true cause for the phenomena, and is probably also a sufficient one. The bronchial tubes, under the conditions given in the cases related above, become unable to empty themselves of their contents, and the pulmonary capillaries also become equally unable to transmit blood through them. The bronchial tubes and in a higher ratio the smaller tubes, become dilated, the pulmonary tissue congested, and exudation (hepatization, or purulent infiltration) follows. These are inflammatory changes in the strictest use of the word, though Majendie, in the quotation given above, criticises the expression. And it is the more important to fully recognise the inflammatory nature of these changes, since we are still too apt to think of inflammation as essentially a nutritive activity in the way of excess—as an "effort" of nature rather then as a "lapse" or paralysis of her powers.

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[1] Op. cit., p. 204.

Besides their pathology, these cases present points of interest in diagnosis. In the first case, the aneurism by compressing the recurrent branch as well as the trunk of the pneumogastric nerve, produced both laryngeal and pulmonary symptoms; in the second case, the conditions were obscured by the absence of respiration over a considerable part of the condensed lung; and in the third, it was from this cause impossible to make a diagnosis by physical examination only. It is one of the evils of a too exclusively humoral pathology that it leads us to overlook the minute anatomical relations of disease, which are in themselves often a key to the sequence of morbid changes. These cases illustrate this proposition, for the possible local effects on the lung of injury of the pneumogastric and pulmonary plexus being recognised, whenever cause sufficient for that injury exists we may anticipate its results, and are not wholly dependent upon physical examination, as we are if we limit our pathological view to the mere changes in the lung, without considering how they are produced.

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[1] Op. cit.

The chief point worthy of note in the physical diagnosis is, that when the paralysed bronchi become choked, the respiration in the part is greatly enfeebled, if not altogether absent, and hence there are no indications from the entrance of the air of the state of the pulmonary tissue. There may be dulness on percussion, absence of vocal resonance and respiratory murmur, and immobility of the chest, not because there is effusion of fluid or some cancerous or other growth, but because the bronchi are obstructed and immobile and the tissue of the lung consolidated.

SECTION IV.

DISEASES OF THE DIGESTIVE SYSTEM.

FATTY STOOLS FROM DISEASE OF THE MESENTERIC GLANDS.[1]

The digestion and absorption of fatty matters claim more than usual attention at a time when these substances are considered as having an especial value in the treatment of strumous affections. It is therefore remarkable that the evacuations are so little studied as a means of informing us to what extent cod-liver oil and the like remedies are absorbed.

With the healthiest digestive organs there is a somewhat definite limit to the absorption of fat, the excess of it in the food passsing away in the evacuations; this limit is obviously much narrowed when the mucous membrane of the small intestine is diseased, or where the mesenteric glands prevent the onward passage of the chyle.

With their imperfect means of observation, the older physicians often surpassed us in their knowledge of symptoms; and though their pathology, from the want of chemistry and the microscope, had a vague expression about it, it was more or less true to nature. *Diarrhœa chylosa* and *Fluxus cœliacus* were recognised by them, and attributed to some impediment in the absorbent system, whereby the chyle was left to escape with the fæces, an opinion which appears to be well founded, though it is probable they often mistook inflammatory exudation for chyle. Modern authors have passed over the subject, or have treated it lightly.

The normal absorption of fatty matters is prevented from two causes; either from a defect in the digestive and emulsifying process, or from disease of the absorbent system. The instances of fatty stools from disease of the pancreas and duodenum, as described by Dr. Bright and others, belong to the former, and are characterised by the fat passing from the intestines, more or less separate from the general mass of the fæces, and concreting upon them; but in the latter case, where the lesion is in the absorbent system, the fat, *being emulsified, becomes incorporated with the evacuation, and is consequently not so easily recognised.* If, however, there be with defective absorption, an inflammatory condition of the mucous membrane and diarrhœa, the oily matters rise to the surface of the evacuations as a creamy film, and produce the pale, chalky, and soapy appearance so characteristic of chronic muco-enteritis and mesenteric disease.

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[1] Reprinted from the 'Guy's Hospital Reports,' vol. i, 1855, p. 369.

In the following case the mesenteric glands were enlarged from strumous deposit; the appetite was voracious, and the emaciation, as usual, extreme. The oily matters of the food passed freely from the intestines, but being blended with the fæces, were not so readily recognised as in fatty stools from disease of the pancreas. Cod-liver oil was administered in small doses experimentally, and passed through the intestines but little changed. The evacuations were for the most part semi-solid. When broken up by a stream of water, and allowed to stand, the oily matter separated as an alkaline soapy cream upon the surface. Under the microscope this consisted of large globules of oil, finely divided oily particles, granular matter, and inflammatory exudation. A quantitative analysis of the fat in the fæces, without a similar one of the food, would have been valueless; for the digestive powers being weak, and the appetite great, the bulk of the fæces was disproportionately large, and consequently any estimate of the mere percentage of fat they contained fallacious.

Reported by MR. HALL.

Henry O—, æt. 13, a delicate child, with light hair and fair complexion, came under treatment in the Clinical Ward, June 8th, 1855. He had been losing strength and gradually emaciating since the beginning of the year, and now presented the appearance of advanced mesenteric disease. Pulse

100, small and feeble. Tongue glazed and red. Appetite very great. Stools loose, and of a light colour. Urine pale, without albumen. Frequent cough without expectoration. Dulness on percussion below the left scapula, with gurgling and pectoriloquy. In the left infra-mammary region the breathing defective, at other parts puerile. The abdomen distended with flatus, and enlarged mesenteric glands can be felt in the umbilical and hypogastric regions. The bowels were generally moved three times in twenty-four hours. The evacuations were pultaceous or liquid, of a dull chalky colour, frothing like soap when a stream of water was poured on them. Under the microscope they were seen to contain muscular fibre in different stages of disintegration, starch cells, &c., and finely divided oily and granular matter like chyle, and inflammatory exudation. Shaken with ether, a large amount of fat separated. *The cod-liver oil administered was readily obtained from the evacuations, so as to be recognised by its colour and odour, after being exposed to the air and losing the fæcal smell.* The appetite was voracious, the emaciation extreme. He died 15th July.

Sectio cadaveris.—In the left chest twelve ounces of sero-purulent effusion. The lung adherent to the ribs at the base of the upper lobe, the free surface covered with a thick layer of rather recent fibrinous exudation. In the compressed lung, at the lower part, several vomicæ, one as large as a pigeon's egg. At the left apex two smaller ones. The right lung crepitant but congested; near the apex a small vomica. Heart normal in structure but extremely wasted. The mesenteric glands much enlarged, and universally affected with strumous disease. The intestinal mucous membrane, from the commencement of the jejunum to the rectum, dotted over with patches of ulceration, with here and there prominent masses of strumous deposit under the surface. The appendix vermiformis was ulcerated through, and the two portions adherent to the cæcum. Pancreas and duodenum healthy. Liver and kidneys healthy.

Although, from the extent of disease in the glands and the mucous membrane, this case may be considered as an extreme instance of the class it is intended to illustrate, it is not the less valuable for the purpose of drawing attention to similar conditions existing in a less degree. It must be of daily occurrence in the treatment of phthisis and other strumous affections, that cod-liver oil fails in doing any good from a morbid state of the mucous membrane of the small intestine, and of the glands of the mesentery; yet its routine administration is persisted in, and a means in itself most valuable is prejudiced, through the carelessness and oversight of those who employ it. If it be ever admissible to limit the attention to one organ in treating disease, it is so in strumous affections, where, as all will admit, the condition of the gastro-intestinal membrane is of paramount importance; yet our remedies at present rarely extend beyond bitters, and alkalies, and cod-liver oil, together with such general palliatives for the cough as naturally suggest themselves.

The disproportionate frequency of tubercular deposit in the intestinal mucous membrane, the mesenteric glands, and the lungs, which organs lie directly in the course of the newly assimilated material, points with no doubtful sign *to the defective constitution of the chyle,* as having an important influence in such a result. Though this view of the subject is generally entertained, and even by some, as by Dr. Hughes Bennett, much insisted on, it is curious to observe that writers make no mention of the character of the evacuations, from the study of which alone can any opinion be formed as to the condition of the digestive and absorbent functions.

CASE

OF

PROBABLE THROMBOSIS OF SUPERIOR MESENTERIC VEIN

AND

RENAL VEINS.

DETACHMENT OF SEVERAL VALVULÆ CONNIVENTES OF JEJUNUM. RECOVERY.[1]

THERE are points in the following case which are in my experience unique.

It was one of copious intestinal hæmorrhage, without fever, followed after some days by the evacuation of portions, more or less complete, of a dozen or more of the valvulæ conniventes (see Plate XX). The patient recovered, and after a period of four years is now in what appears to be good health, and at least without any further intestinal symptoms.

On the 12th of October, 1879, I was requested by my friend Mr. Venning to see this case with him, in conjunction with Mr. Worship, of Sevenoaks. The patient was a young gentleman, æt. 23, suddenly suffering from very profuse intestinal hæmorrhage. The onset of the symptoms was unexpected, and began with *very severe pain in the abdomen,* which appeared to be relieved by a warm bath; and then followed the hæmorrhage. There was slight sickness, but no hæmatemesis. The patient was apparently well up to the sudden attack of pain, eating and drinking as usual, only that some days before there had been some slight and transient œdema of the right hand and part of the forearm, which could not well be accounted for, and which had disappeared at the time of the attack. The œdema was vaguely though probably truly referred to a secondary taint, a primary sore having been contracted five months before; but secondary spots on the skin were doubtful.

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[1] Reprinted from the 'Guy's Hospital Reports,' vol. xxvii, 1884, p. 15.

On passing the intestinal evacuation of blood from one vessel to another, it was noticeable that it varied in colour, from the darkest venous with black coagula, to that of a lighter tint, indicating that the bleeding surface was extensive and high up in the intestine. The warm bath appeared to have given great relief, and the patient expressed himself as feeling as if something had been set free by it, but this relief was probably due to the hæmorrhage.

The question which naturally engaged the attention of my colleagues and myself was, as to the pathological cause. There had been no fever, no previous illness, and nothing complained of but the slight transient œdema mentioned as occurring a few days before. The conclusion was, that the cause was mechanical, and one that obstructed the mesenteric vein high up. The severe pain, the bleeding, and the character of the blood favoured this opinion as to the seat of the obstruction. It was conjectured, also, that the obstructing cause was outside the vessel, and might be a gummatous deposit. Whether it were so or not cannot be settled, though the subsequent history supported this opinion. Assuming it to be so, it was to be expected that other veins in the neighbourhood, as the renal veins, would suffer; and this subsequently occurred. The urine at the time of the attack was carefully examined. It was pale, sp. gr. 1015, slightly opalescent by heat and nitric acid.

On the night of the 15th, three days after the attack, the patient fell into a state of sudden collapse, and became almost pulseless. The following day the temperature was 100·8°, pulse 108, abdomen tympanitic, constant vomiting, ejecta bilious without blood, great thirst. On the 17th symptoms less urgent, urine with even less evidence of albumen, temperature 99·4°, pulse 90. On the 18th pulse 84, no sickness, evacuations bilious and without blood; the two previous days they had been melæuous. Urine darker in colour, sp. gr. 1020, a large amount of albumen. On the 20th and 21st, sloughs of valvulæ conniventes were evacuated, but with no trace of muscle tissue attached to them. From this day to the 27th, portions of mucous membrane, rings or portions of rings (valvulæ conniventes), were passed in the evacuations, the free surfaces being rich in villi. On the 27th the last of these sloughs came away. The patient was going on well, abdomen soft, evacuations formed and natural in appearance. There were no external swellings, or nodes, or œdema in any part of the body. The renal secretion, however, continued to undergo remarkable changes, though the patient was otherwise convalescent. On November 7th, urine acid, of a dull amber-greenish colour, sp. gr. 1025, one quarter to one half its volume of albumen deposited on boiling; casts with hæmatin, and exudation cells; pure hyaline casts; small separate concretions of crystalline hæmatin.

November 21st.—Urine acid, depositing urates and a peculiar clot of loose translucent bloody mucus, loaded with albumen, sp. gr. 1031; after separating albumen by boiling, sp. gr. 1025. There had been no further intestinal symptoms since the 27th of October.

On the 17th of January of the year following (1880) the same abnormal state of the urine continued: sp. gr. normal, much albumen, and still a deposit of the same peculiar bloody mucus. This was suspected to come from the pelvis of the right (?) kidney. Notwithstanding this condition of the urine, the patient was convalescent, appeared to be in his usual health, and has so continued up to the present time, November, 1883. He has been able to travel and otherwise enjoy himself in his usual manner like a healthy person, but the urine continues to be albuminous, though of normal sp. gr., and free from the loose translucent bloody mucus, which occurred three years ago.

In the sudden onset of pain the case accords with what is known of sudden distension of vessels, whether from thrombosis or dissecting aneurism. Clinically, this fact of sudden and severe pain in the abdomen due to sudden distension of vessels has to be borne in mind in diagnosis, so as to separate the cases from attacks of ordinary colic, the pain of gall-stones and of renal calculus. As the patient whose case is here given recovered, and is now apparently in health, the pathological history is happily defective, but it may in part be supplemented by what occurred in a case recorded by the late Dr. Hilton Fagge.[1]

A lady æt. 34, of pale and sallow complexion, and having a slight scar in the lumbar region, from an abscess (?) in childhood, had given birth to a healthy child a month previously; and was suddenly seized, at 6 a.m., with violent pain in the abdomen and vomiting of a rather viscid blood-stained fluid; symptoms of collapse; abdomen flaccid; no tenderness anywhere, nor unnatural fulness. "I could not," says Dr. Fagge, "form the slightest idea as to the cause of this sudden illness. Patient died at 5 o'clock p.m., eleven hours from the beginning of the attack."

A post-mortem examination was held the following day. "When the abdomen was opened the first thing that attracted observation was extreme congestion of part of the small intestine. The congestion began about four inches from the termination of the duodenum, and ended at about the middle of the small intestine. The internal surface of the bowel was deeply reddened, and covered with shreds of mucus, so that it seemed as though the mucous membrane itself was being detached. The congested state of the jejunum was so like what would have resulted from internal strangulation of the bowel that I searched very carefully for any hernia or other cause of obstruction. But none could be found. Moreover the affected part of the intestine was by no means distended; it was not larger than the rest. The real explanation of the state of the intestine, however, became apparent when we cut through the mesentery to remove it. The branches of vein coming from the affected part were found to be distended with adherent coagulum. This condition was traced up the superior mesenteric vein into the trunk of the vena portæ. The rest of the abdominal viscera were apparently healthy. The vena cava had by no means been in a normal condition before the thrombosis began. It was flattened and narrowed, and embedded in a quantity of very firm fibrous tissue. This induration extended downwards along the brim of the pelvis and down the sides of the lumbar vertebræ. The intervertebral discs were all healthy, and the interior of the bodies of the vertebræ appeared perfectly normal. But the left side of the last lumbar vertebra seemed to be slightly excavated, and on scraping it with the knife I found that it felt rather rough, and that I could cut away some portions of osseous substance embedded in the tough fibrous tissue." [1]

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[1] 'Pathological Transactions,' vol. xxvii, 1876, p. 124.

This case of Dr. Fagge's probably supplies the evidence wanting in that I have here recorded. The evidence of an old affection of the connective tissue around the vessels in the abdomen, the pale and sallow complexion of the patient, and the old scar in the lumbar region show an old-standing cachexia; and there being no mention of tubercle, this cachexia may probably have been due to an inherited taint of syphilis. If so it would further accord with the history of the case given above, in which there was a plain history of taint though no external evidence of tertiary effects. Without laying undue weight as to the parallelism of the two cases as respects their latent pathology, it will be admitted to be a question to be raised in similar instances how far a syphilitic cachexia, affecting the connective tissue (gummata) around vessels, and causing hæmorrhage, is not to be placed side by side with disease of the connective tissue around nerve-trunks from the same cause. In the case here given my colleagues and myself agreed to treat the case as one of this kind, and prescribed the iodide of potassium and bichloride of mercury, notwithstanding the highly albuminous state of the urine; and the least that can be said is that the patient is well, except for what may possibly be referred to one (?) of the kidneys. It further seemed to us probable that only one kidney had suffered from the venous obstruction, and this one severely, as shown by the state of the urine, and especially by the deposit in it of the peculiar loose, bloody, translucent mucus which has been mentioned, and which we attributed, rightly or wrongly, to changes in the renal pelvis. The affection of one kidney alone is suggested by the course of the case, and this would be compatible with the patient's good health, although the renal secretion has never recovered its normal state.

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[1] Dr. Frederick Taylor has also brought before the Pathological Society ('Transactions,' vol. xxxii, 1881, p. 61) a case of a somewhat similar kind. A child, æt. 5, was suddenly seized one afternoon with abdominal pain and vomiting; she had violent paroxysms of pain all night, and died the following morning. On post-mortem examination blood was found effused into the peritoneal cavity, and into the cavity of the small intestine. The jejunum and ileum were deep red in colour. The superior mesenteric artery was found obstructed by being matted together with a fibrous mass in the mesentery; and the mesenteric vein contained a thrombus at the same point.

In respect of the sloughed valvulæ conniventes it is to be remarked that the sloughs differed entirely from what would have been passed in intussusception; they were unaccompanied by any trace of muscular coat. What really occurred is probably explained by the post-mortem in Dr. Fagge's case. He states that at the affected part it seemed "*as though the mucous membrane itself was being detached*;" and this occurred in the case here recorded, as shown by the detached valvulæ conniventes thrown off in broken portions. This case suggests some thoughts as to therapeutics, —the uselessness, if not harmfulness, of trusting symptoms. The profuse hæmorrhage would have seemed to call for styptics, yet the hæmorrhage was useful and curative, and drugs to stop it would have done nothing but harm. The hæmorrhage was a necessity of the conditions, and therapeutical. On the question of artificial depletion it supplies some lessons. Bleeding did good in this case at least.

I am indebted to Dr. Charles Hood for separating the sloughs of mucous membrane from the fluid in which they were passed, and also for arranging them as seen in the drawing (Plate XX).

Report by Dr. Goodhart.—The specimen as preserved in the bottle and extended upon talc is ten and a half inches long, but there are many gaps in this length to reduce its actual measurement. It consists of alternating thin and thick bands of membrane, the latter corresponding no doubt to the valvulæ conniventes of the intestine, and the thin part to the mucous membrane between them. Those parts which are thicker, but all more or less, have the velvety appearance characteristic of the villous surface of the intestine. Some are strips of membrane representing half the circumference of the small intestine, others more, and many are complete rings of the entire circumference of the bowel.

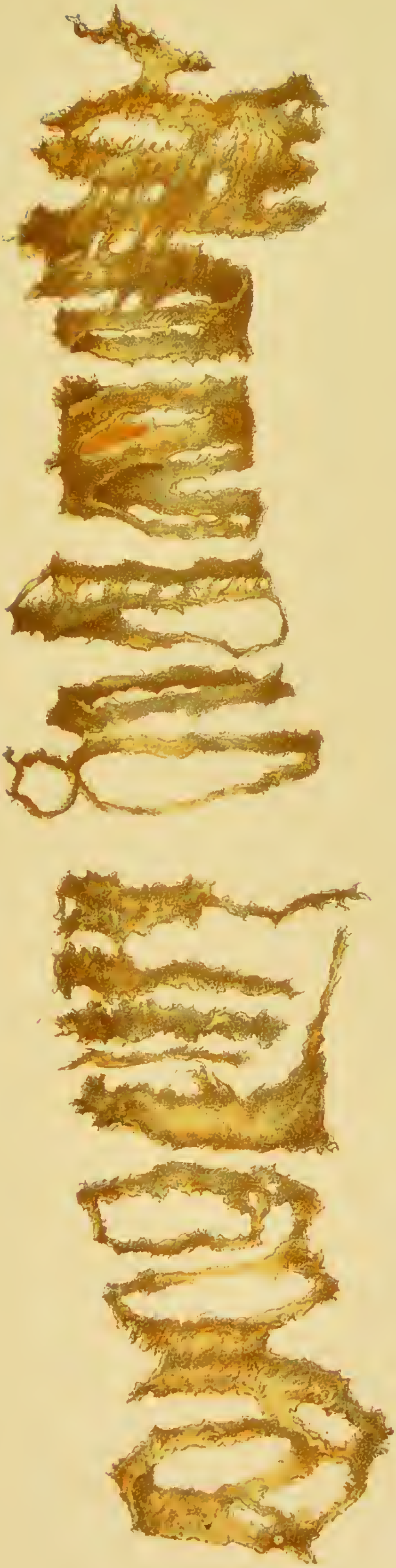
No parallel bands of muscular fibre can be detected on the closest examination and by transmitted light.

There can be no doubt that a localised enteritis and necrosis of the mucous membrane has occurred, and that the mucous membrane has separated in consequence. But it is remarkable that the disease should have been at the same time so severe, and yet so superficial that the muscular coat is entirely spared.

DESCRIPTION OF PLATE XX.

Coils of valvulæ conniventes passed in the evacuations, the inner surface velvety from villi.

Plate XX



West, Newman chromo.

SECTION V.

RHEUMATIC FEVER.

CASES OF RHEUMATIC FEVER,

TREATED FOR THE MOST PART BY MINT WATER.[1]

In studying the treatment of acute disease it is impossible not to observe how confidently remedies, various and even opposite in their modes of action, have been recommended; the experience of physicians, of justly acknowledged great reputation, quoted; and results shown which in many cases appeared by no means unsatisfactory.

It is probably while endeavouring to explain the effects, so little to be expected from such different kinds of treatment, an opinion has been gradually formed that the natural course of the disease had more to do with the result than the remedy.

With little doubt, however, we shall all agree in acknowledging that any plan of treatment having for its object to show when, and how far it is necessary to have recourse to the aid of medicine in the treatment of rheumatic fever, is entitled to our utmost consideration. Therefore, cases treated, as the following cases have been, by such simple means that we might almost consider them to have been unassisted by any remedy, are invested with no little interest. And although the collective results may not warrant us condemning or praising any particular kind of treatment, yet they will probably warrant us concluding that we ought not to be too hasty in considering the apparent sudden and favorable change in the symptoms due to any medicine administered.

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[1] Collected from the Clinical Books of Dr. Gull, with some remarks on the natural history of that disease. By Henry G. Sutton, M.B.Lond. Reprinted from the 'Guy's Hospital Reports,' 1865, p. 392.

We have more than once heard Dr. Gull remark that these cases cannot be considered to have been treated solely on the expectant plan, for an occasional dose of Dover's powder, or half a grain of opium, night and morning, and two or three ounces of brandy a day, are remedies that might be fairly expected to exercise some, although perhaps little, influence over the course of the disease.

One of the chief objects Dr. Gull had in view in instituting these investigations was to endeavour to gain a more accurate knowledge of the course the disease tends to take —that is, its natural history; and while, no doubt, we are enabled to compare cases in which little or no medicine has been given, with others in which it has been administered, and arrive at some conclusion, yet we cannot forget that rest in bed, a well-regulated diet, and good nursing, are powerful elements in the treatment of any acute disease,—so powerful, in fact, that experience has shown not a few cases of continued fever and of pneumonia, and of other acute diseases, recover without further assistance.

Before proceeding to give records of these cases, we would beg to mention that no selection was made, but that Dr. Gull treated the cases which happened to be admitted into his wards on the same plan; and we would further beg to say that these reports were not kept for any special object, nor are they as complete as they might be; yet the facts stated may be fully relied upon, and so far answer our purpose.

A severe case of rheumatic fever; pericarditis; endocarditis.

George E—, æt. 24, a groom, admitted June 5th, 1863, into Stephen Ward; temperate habits; enjoyed good health until lately. June 2nd, had cold chills, and pains in his joints; did not at the time feel particularly ill, but gradually got worse and worse. On admission, said that this is his first attack. Pain with effusion into both knee-joints; pulse 104; respiration tranquil. A soft systolic murmur is heard. Area of cardiac dulness normal. A shooting pain in the epigastrium. Says that he is a little troubled with wind on the stomach. Tongue white, slightly dry, and brown towards the tip. Julep. Menthæ et Ext. Taraxaci ℥j, Æther. Chlor. ♏x, in singulis dosibus ter die. Milk diet. Brandy ℥j per diem.

June 6th.—A delicate to-and-fro exocardial murmur, heard chiefly on a level with, also an inch below, the left nipple. Over the second left costal cartilage a slight murmur heard, synchronous with the first sound; he makes no complaint, except of pain across the epigastrium. Breathing tranquil, 20 in the minute; pulse 96. On drawing a deep breath, feels a sharp pain between the fifth and sixth ribs over the apex of the heart.

8th.—Pulse 72. Has more pain in chest at night than during the day. Rept. mist.

9th.—Can move his legs up and down the bed. Tongue furred, red at the tip; to-and-fro sound heard over the heart; breathing tranquil; *joints free from pain.* Rept. mist.

12th.—To-and-fro sound much the same, but now heard all over the cardiac region. No increased dulness; *joints free from swelling* and pain; skin cool. Rept. mist. Diet the same.

16th.—Friction sound still remains, but heard very softly. Pulse 76. Rept. mist.

18th.—Feels better; in no pain.

19th.—Pericardial sounds cannot be heard; cardiac dulness normal, but there is a soft systolic bruit at the apex; pulse 100; joints free from pain; urine pale amber colour. Rept. mist. Milk diet and two eggs.

25th.—Got up to-day for the first time. Pulse 92.

28th.—Steadily improving.

July 3rd.—Under the nipple, and over the apex of the heart, a systolic bruit is heard, and it is slightly musical.

6th.—Went out of the hospital.

Severe case of rheumatic fever; myocarditis.

John G—, æt. 30, a horse dealer, admitted June 6th. Has been in the habit of drinking a good deal of beer—fourteen or fifteen glasses a day. Seldom ever drank spirits, and said that he never felt drunk. His occupation compels him to walk a great deal, and while walking usually perspired very freely. Said that he had always enjoyed very good health previous to his present illness, that he had always enjoyed his meals, but never could eat fat meat. In the morning after drinking, occasionally suffered with what he called a "cough," and not unfrequently he vomited. Physiognomical appearances:—Well-developed head; hair dark brown, inclined to be thin; complexion florid; features regular; alæ nasi moderately, upper lip somewhat thick; eyes dark brown; his teeth were sound, regular, and worn down at the edges; his skin was fair and thin; height five feet ten inches; weight thirteen stone; he is a muscular man, and says that he was very fat twelve months ago.

The supposed cause of his illness he believes to have been sleeping in a damp bed three weeks ago. Two or three days after doing so he began to have pain in the limbs, and, as he expressed it, "felt very bad." Obliged to go to bed, and soon afterwards he came into the hospital. He further stated that eighteen months before this attack he had suffered now and then with pain in the shoulders and neck, but his joints were not swollen.

On admission he complained very much of severe pain in both his wrists and his knees. Skin hot, and perspiring very freely, with a strongly marked rheumatic odour; tongue white and thickly furred; pulse rather quick and feeble; area of cardiac dulness normal, sounds also, except that the first sound is somewhat feeble. Opii gr. j statim. Dec. Lini pro potu. Milk diet.

June 9th.—Much the same, in great pain, and the joints swollen. Skin hot, and perspiring profusely; bowels confined. Ol. Ricini ℥ss; Opii gr. j o. n.

11th.—Says the pain in the joints is no better; the ankles also affected. He appears low and depressed. Skin hot; cannot move in bed; heart's sounds without murmur, but the first sound is exceedingly feeble; respiration quick. Pil. Hydrarg. gr. v, hâc nocte; et pergat., P. Sodæ Potassio Tart. efferves., P. L. ex aq. tepid, cras primo mane si opus sit. Julep Menthæ et Taraxaci ter die. Brandy one ounce three times a day in barley water.

Dr. Gull here remarked, although there is here no bruit, and no markedly increased dulness, yet the peculiar, feeble, almost suppressed first sound, probably indicates that the muscle of his heart is mainly suffering.

14th.—Much the same. In great pain in his limbs, and also complains of pain and oppression in his left chest. Ordered Emp. Canth. to be applied to the chest. Pulv. Opii gr. iss in pil. o. n. Rept. mist.

22nd.—States that he feels better, not in so much pain; tongue clean, largely indented at the edges; pulse feeble; cardiac dulness normal; impulse feeble, but discernible. First sound at the apex is now prolonged almost into a murmur; doubling of the second sound; rhythm of the heart irregular. Rept. Pil. and mist. Two eggs. Rept. brandy.

25th.—Improving; heart's action much the same; pain much easier.

28th.—Still improving. First sound still prolonged; rhythm of the heart regular; pain going away. Rept. mist. Middle diet.

July 1st.—Much better. In no pain. Decoct. Cinchonæ c. Soda ter die.

7th.—In pain all over his body. Skin hot and perspiring; first sound markedly prolonged at the apex, but could not be called a bruit. Complains of pain in the chest. Emp. Canth. pect.

9th.—Relieved by the blister; not in so much pain.

11th.—Still more free from pain. Rept. mist.

14th.—Free from pain; skin cool; tongue clean; appetite good.

16th.—Discharged cured. When he left the hospital, although there was no decided bruit, yet the first sound at the apex was unduly prolonged, and almost a murmur.

Case of rheumatic fever; pericarditis; albuminous urine.

John S—, æt. 22, admitted October 29th, 1864; always been rather delicate. Three years ago had rheumatic fever, and laid up two months. Ten months ago had another attack, which lasted three weeks. Further said, that three weeks since he suffered very much with faceache, and continued to do so for a week; at the end of that time he began to suffer with pains in the limbs, and was soon compelled to take to his bed.

October 29th.—When admitted his skin was very moist, and his countenance anxious; pupils rather dilated; tongue furred; pulse 132; resp. 40. Over the heart a loud to-and-fro pericardial murmur heard. Has great pain in all his limbs, but no swelling. Can move his limbs, but it causes him great pain. Has the rheumatic odour. Urine sp. gr. 1025.

30th.—Ordered Haust. Menthæ et Taraxaci ter die.

31st.—Countenance anxious; complains of great pain in the chest; has not slept for two nights; bowels confined; legs much better, but his arms are still very painful; skin perspiring, and has the rheumatic odour. Urine sp. gr. 1027, acid reaction, and highly albuminous. Pulse 120, resp. 50.

November 1st.—Was ordered a grain of opium last night, which caused sleep; pain in the chest better; pain in the left knee. Pulse 120, resp. 48. Urine sp. gr. 1026, albuminous.

2nd.—Pulse 118, resp. 40. Pericardial rub not so distinctly heard.

3rd.—Pulse 120, resp. 50. Countenance anxious; arms very painful.

4th.—Little better, can put his hand to his head; did not sleep last night at all. Pulse 112, resp. 48. Is not perspiring so much. Urine thick and amber colour; sp. gr. 1027, slightly albuminous.

5th.—Can move his hands and arms much better; pericardial rub scarcely perceptible. Pulse 120, resp. 54.

6th.—Has an eruption of sudamina over the abdomen; the contents of some of the vesicles are translucent and acid to test-paper, the contents of others are opaque and alkaline. Pulse 120, resp. 48.

7th.—Pulse 112, resp. 44.

8th.—Can scarcely detect the contents of any of the sudamina to be alkaline, but they are disappearing. Pulse 115, resp. 42.

9th.—The eruption has entirely disappeared; seems much better, but his hands continue tender. Pulse 120, resp. 48.

10th.—Perspiration on the face gives an acid reaction, but only slightly so. Pulse 124, resp. 44.

11th.—Seems much better; he has a loud murmur at the apex. Pulse 120, resp. 40.

13th.—Much better; appetite returning; sleeps better at night. Pulse 82, resp. 16.

15th.—Does not sweat at all; in no pain. Pulse 90, resp. 22. Urine albuminous; sp. gr. 1024.

Went on improving up to the 25th, when he went out.

Rheumatic fever, second attack; systolic bruit on admission.

William R—, æt. 24, carman, admitted November 23rd, 1864, into Stephen Ward. A strong, healthy-looking man. Seven years ago had rheumatic fever, and laid up six or seven weeks with it. Ill ten days before admission. His joints are generally painful but not swollen, and other well-marked general symptoms. First sound of the heart attended with a slight systolic murmur. The area of cardiac dulness is oval-shaped, two inches in width and about two and a half inches in length. Pulse 120; urine sp. gr. 1030; no sugar or albumen. Julep. Menthæ et Taraxaci ℥j ter die.

November 29th.—Pains in the limbs gone. Pulse 74; tongue clean.

30th.—All the rheumatic symptoms have disappeared.

December 2nd.—Dr. Gull saw him to-day, and said that the bruit is no longer heard, and that he is much better.

4th.—Convalescent. Potassio Ferri Tartrate gr. xv, Ex. Julep. Menthæ ter die. Middle diet.

10th.—Gone out cured.

Rheumatic fever; pericarditis with effusion.

Henry J—, æt. 26, married, a firewood-cutter, admitted October 15th, 1864, into Stephen Ward. A thin, pale, weakly-looking man. Has been ill fourteen days, not confined to his bed, but not able to work. Pain and swelling of the hand. Perspires a little at night.

October 15th.—Julep. Menthæ c. tarax. 6tis horis; Extr. Opii gr. ss, nocte maneque si opus sit. Milk diet. Brandy ℥ij per diem.

19th.—Pulse 84. Feels better. Wrists and fingers not so stiff and painful. Does not sweat so much; tongue white and furred; systolic bruit at the base of the heart; no friction sound; cardiac sounds distant; increased dulness over the cardiac region. Rept. Mist.

22nd.—In a good deal of pain; passed a very bad night. Rept. Mist.

25th.—Not much improved; still in great pain; area of pericardial dulness increased. Ol. Ricini ℥ss statim.

26th.—In less pain; dulness over the heart much the same.

27th.—Increased pain in the right wrist. Rept. Mist.

28th.—In no pain; sweats less; area of cardiac dulness now natural.

November 3rd.—Not so well; pain in his left shoulder; perspiration returned again; has severe pain in his left shoulder. Pulse 120. Passed a bad night.

4th.—Much better to-day. Pulse 104. Haust. Quiniæ ter die.

8th.—Much improved the last three or four days. Pulse 76; tongue clean; free from pain. After this he gradually got well, and left the hospital.

28th.—Quite recovered.

Rheumatic fever with aortic regurgitation.

Thomas F—, æt. 11, admitted March 2nd, 1864, into Clinical Ward, under the care of Dr. Gull. Says that he has had two previous attacks, the first when he was five years old, the next three months ago; since which time he has never been so strong as he was previous to the second attack. His present illness commenced ten days ago, when he got wet feet, and the same day felt pain in the joints, headache, thirst, and general feverishness. The joints did not swell much, but were extremely painful.

On admission complains very much of pain, especially in the knees and ankles, which are hot and a little swollen. His tongue is red and rather dry, and in parts covered with white fur. Pulse 100, full and sharp; skin dry and hot; complains of great pain; urine scanty and high coloured; bowels constipated. A double bruit heard over the region of the heart.

Ordered—

℞ Hydr. c. Cretâ gr. ij, statim ;
Ol. Ricini ʒj, post horas quatuor.
Milk diet with brandy ℥j per diem.
Ext. Taraxaci ℈j ;
Ext. Julep. Menthæ ℥ss, 6tis horis.

March 3rd.—Has had a restless night, complains of pain all over him, and of headache, and of being very thirsty; sweating freely; skin hot; pulse 100; bowels acted twice; knees less painful.

4th.—Swelling and pain in the knees have subsided, but now both wrists are affected, the left especially. Did not get much rest during last night. His skin is hot and dry; tongue is red; lips cracked; great thirst; no appetite. About noon he began to perspire very freely, and felt easier all the rest of the day. Pulse 120. Complains of pain over the cardiac region; heart's action is excited. The double murmur appears to be endocardial, and due to aortic regurgitation, the result of previous attacks of this disease. Rept. mist.; brandy ℥iss per diem.

The pain only in left wrist, not severe; tongue clean and not red; skin dry but not hot; bowels acted freely this morning; urine passed more abundantly, contains a large quantity of urates. Heart's action much quieter; pulse 100.

6th.—*Free from pain.* Pulse 90. Slept well all last night. Appetite is returning.

8th.—Quite free from pain in the joints, but complains of headache. Pulse 90; skin cool; tongue clean and moist; bowels acted daily; urine clearer.

10th.—Felt slight pain in his wrists again yesterday and to-day, but they are not swollen, although hot and a little red; appetite better, and he sleeps well.

13th.—Going on well.

15th.—Allowed to get up for a few hours, as he is getting restless and tired of bed. Rept. Mist.

17th.—Sitting up quite free from pain. His pulse is 100 in the erect posture; bowels open once a day; he feels pretty well.

19th.—Complains of pain and aching in his back towards evening, and of being rather thirsty; tongue clean; pulse 100.

20th.—Does not feel quite so well to-day; skin hot; pains in the back and loins; tongue rather red and dry; no appetite.

21st.—His skin is hot and dry; complains of headache; his wrists are swollen; pulse 140. Continue Taraxacum mixture; brandy ℥iij in 24 hours.

23rd.—A little easier. Pulse 120; heart not so irritable; he is still in pain; tongue much the same.

25th.—In less pain. Tongue not so red, but still furred; pulse 120. He does not sleep much. Bowels acted naturally; urine high coloured, deposits lithates in small amount.

28th.—Better again. Pulse 100. Not in any pain; tongue cleaner; skin perspiring and cool.

30th.—Improving.

31st.—Went home relieved.

Rheumatic fever; prolonged first sound; irregular rhythm of the heart.

George S—, æt. 19, a barman, admitted June 17th, 1863, into Stephen Ward. Has enjoyed good health, though a "strumons" looking subject. Four days ago awoke in the morning and found all his joints stiff and swollen, but not very painful.

19th.—Heart's sound free from murmur, but the first sound prolonged. The pulse jerking; knee-joints much swollen, also the left wrist; skin perspiring. Pulv. Doveri gr. v, o. n.; Julep. Menthæ et Taraxaci ter die. Milk diet.

23rd.—First sound much clearer; no bruit heard. Rept. Mist.

24th.—Rhythm of the heart irregular; no bruit heard; *joints free from pain;* skin cool. Rept. Mist.

25th.—Rhythm still irregular; no bruit; tongue moist.

29th.—Much better; out of bed.

This case was only reported up to this point.

Charles A—, æt. 21, admitted May 3rd, 1863, into Stephen Ward. Ill three days before admission.

May 3rd.—Swelling of the left wrist; tongue furred; pulse 100; bowels constipated. Hyd. c. creta gr. v, stat. Pulv. Seidlitz post hor. quat.; Julep. Menthæ et Ext. Taraxaci ter die. Milk diet. Brandy ℥iij per diem.

7th.—Tongue dry and brown. Rept. Mist.

10th.—Much the same; still in pain. Rept. Mist.

13th.—Pain came on in the right knee. Rept. Mist.

14th.—Pain in both knees, not swollen. Rept. Mist.

19th.—*Free from all pain;* no swelling of any of the joints.

23rd.—Much better. Decoct. Cinchonæ et Sodæ bis die. Full diet.

June 4th.—Discharged.

James G— (age not given), admitted November 11th, 1863, into Stephen Ward. Never had any illness; appetite generally good; had drank freely of spirits; severe pain in the joints, especially in the ankles.

November 13th.—Much the same. Julep. Menthæ c. Ext. Taraxaci ʒj, 6tis horis. Brandy ℥ij. Milk diet. Pulv. Doveri gr. x, hâc. noct.

15th.—Hands, knees, feet, and shoulders hot, swollen, and painful; skin hot, but not sweating; pulse 92, full; percussion-sounds over the heart normal; first sound of the heart a little feeble, but without murmur; second sound clear. Dr. Gull thought the heart at present free from rheumatic inflammation. Rept. Mist.

December 4th.—Remained much the same; very unwell; still taking the same mixture.

9th.—Improving. Decoct. Cinchonæ, ʒij bis die.

13th.—*He was free from pain.*

Here the report ends.

Rheumatic fever; eighth attack.

Napoleon L—, æt. 23, admitted November 28th. Has been in the hospital seven times with symptoms of rheumatic fever. Six weeks before admission had pain and stiffness in the joints; systolic bruit at the apex. On admission could not move the right leg, but two days afterwards it was nearly well. Perspiration; skin very hot; pulse 92.

December 5th.—Right shoulder and wrist affected, pain only slight; complains, on taking a deep breath, that he has pains across the chest.

12th.—Skin cool; slight pain in the shoulders; feels altogether better.

14th.—Out of bed to-day.

17th.—Had a pain in the loins, and still in pain in the shoulder. After this he gradually improved. Discharged on the 28th.

George G—, æt. 18, waiter, admitted July 18th, 1864. Drinks hard of beer; general health good; present illness began eight days ago; pain, swelling of the joints, and perspiration.

July 18th.—Ol. Ricini statim; Julep. Menthæ c. Ext. Taraxaci ter die.

20th.—A systolic bruit at the apex. Rept. Mist.

August 2nd.—Gets up; *all the pain gone.* Ferri Pot. Tart. gr. v, Ext. Camph. ℥iss ter die.

10th.—Pain in limbs gone.

18th.—Went out well.

Well-marked case of rheumatic fever; endocarditis.

Charles H—, æt. 33, horsekeeper, admitted July 31st, 1863, into Stephen Ward. Intemperate habits. Always enjoyed good health, except having had now and then a cold, until fourteen days ago, when he went to bed quite well, but in the morning was not able to put his right foot to the ground. The right ankle was painful and swollen; next day his left foot was attacked.

July 31st.—His ankle and shoulder-joints are much swollen; tongue moist; pulse 100, very full; skin hot, and sweating freely; first sound of the heart replaced by a soft blowing murmur; second sound clear; heart's visible impulse is seen unusually distinctly. Hyd. c. Cretâ gr. v, stat., et Ol. Ricini cochl. med. post horas quatuor; Ext. Taraxaci ʒj et Julep. Menthæ ℥j ter die. Milk diet.

August 3rd.—Feels much better; hands not so swollen; perspires freely. Rept. Mist.

5th.—Feels better; tongue clean. Rept. Mist. Milk diet.

7th.—Free from pain; skin cool; systolic bruit still remains. Rept. Mist. Milk diet.

11th.—Free from all rheumatic symptoms, excepting the systolic bruit. Rept. Mist.

14th.—Improving. Diet, fish.

17th.—Still continues to improve.

18th.—Ferri Pot. Tart., gr. v, et Julep. Menthæ ter die. Middle diet.

28th.—Discharged.

A well-marked case of rheumatic fever.

John H—, æt. 20, admitted August 3rd, 1864; a rope-maker. Ill eight days before admission.

When admitted, pain, swelling of the joints, and sour perspiration; tongue furred. The first attack. Heart's action healthy.

August 5th.—Julep. Menthæ, Ext. Taraxaci ter die.

7th.—Feels better and stronger.

10th.—Free from pain, except in the right shoulder.

13th.—Convalescent.

22nd.—Discharged cured.

Rheumatic fever, fourth attack; pleurisy.

John B—, æt. 16, admitted August 17th, 1864. Parents healthy. It is the fourth attack. Began two days ago, with pains in the knees and shoulders; pulse 116, systolic bruit at the apex; tongue moist, covered with white fur.

17th.—Pleuritic rub, with tubular breathing over the left side; pericardium seems free; pain in the joints. Julep. Menthæ c. Ext. Taraxaci ter die.

22nd.—Heart's impulse strong; dulness increased; intercostal spaces drawn in during the systole.

24th.—Pain in the left side and pleuritic rub.

30th.—Systolic bruit over the apex.

September 12th.—Rapidly improved; went out well.

Rheumatic fever, well marked; relapse.

John S—, æt. 21, admitted October 27th, 1864. Pain in the joints, swelling; heart normal. Haust. Menthæ c. Taraxaci ter die; brandy and water, hot.

November 7th.—Entirely free from pain; appetite good; urine normal.

12th.—Appears convalescent.

17th.—Been out in the park. The pain returned in his left shoulder.

20th.—Not so severe.

24th.—Again free from pain.

26th.—Left the hospital quite well.

FEMALES.

Rheumatic fever; albuminous urine.

Maria L—, æt. 34, admitted December 5th, 1864, into Mary Ward; married; one child; always had good health; ill a week before admission with supposed rheumatic fever. Suffering very great pain in the joints, the latter also swollen. Urine albuminous; pulse 100; resp. 24.

December 6th.—In very great pain; sweats a great deal; no bruit; urine the same; pulse 102; resp. 26.

7th.—Passed a very restless night; pain most severe; heart's sounds normal.

8th.—In great pain, especially in the shoulder. Dr. Gull saw her, and ordered Julep. Menthæ ℥j, Ext. Taraxaci ʒj ter die.

9th.—Passed a better night, and feels much better; skin cooler; pulse 85; resp. 21. Dr. Gull said she was doing well. Hydrarg. c. Cretâ, gr. v, statim. Ext. Opii gr. j, o. n. s.: Ol. Ricini cochl. med.

10th.—Much better. Urine still albuminous; pain much less.

12th.—Not so well. Pain much worse; skin hot; resp. 22 and difficult; complains of want of sleep.

15th.—Not so well. Urine still albuminous; great pain and sweating; pulse 90; resp. 22. Continued much the same until the 19th, when she appeared much better; less pain; pulse 88; resp. 22.

26th.—Not so well; pain in the knee returned.

January 9th.—Dr. Gull thinks she is going on well.

13th.—Pain in the knees.

20th.—Going on well.

24th.—Much better. No pain anywhere. To go out on 27th.

Rheumatic fever: double pleurisy; endocarditis.

Jane C—, æt. 29, admitted November 18th, 1863, into Mary Ward; charwoman; unmarried; always delicate; a pallid-looking woman, with dark eyes. Her mother suffered with rheumatic gout. This patient stated that five years ago she was in Westminster Hospital for rheumatic fever. Six weeks ago pain in her limbs, obliged to give up work. Four days ago ankles swollen; heart sounds feeble; first sound prolonged; pain in the joints and swelling; perspiration; physical signs of acute pleurisy over bases of both lungs.

November 21st.—Ext. Opii gr. j, o. n.; Julep. Menthæ ℥j; Ext. Taraxaci ʒj ter die. In some pain in the joints; perspiring very much; feeble systolic bruit.

23rd.—Sweating profusely; very acid; dulness and egophony right base; friction over left base.

30th.—In less pain; friction sound distinctly heard over right base.

December 1st.—*Pain gone;* appetite returning.

6th.—Got up to-day.

8th.—Bruit still heard.

10th.—Improving. Ordered Decoct. Cinchonæ ℥j ter die.

13th.—Gets up daily. Went out cured.

Rheumatic fever, third attack.

Elizabeth B—, æt. 25, admitted August 2nd, 1864, into Mary Ward; single; laundress; health usually pretty good. Has had rheumatic fever twice before. Been ill fourteen days before admission with sore throat and headache, believed to be a cold. On admission, joints of the legs only affected, but those of other parts had been affected. Feels very weak; tongue clean; no appetite.

August 2nd.—Haustus Menthæ c. Sodæ bis die.

8th.—*Free from pain;* out of bed.

11th.—Gone out well.

Rheumatic fever, second attack; heart complication.

Emma F—, æt. 15, admitted August 11th, 1864, into Mary Ward. Second attack of rheumatic fever; pain and swelling in the joints, especially at wrists; vertigo; pulse 112.

August 12th.—Julep. Menthæ et Taraxaci ter die; systolic bruit.

24th.—Getting better.

26th.—Improving; sleeps well.

29th.—Systolic bruit gone, and in other respects much better.

30th.—Ferri Cit. c. Quiniæ gr. iv.

31st.—Complains of feeling weak.

September 1st.—Improving.

12th.—Out of bed to-day.

24th.—Went out well.

Rheumatic fever; pericarditis.

Emily W—, æt. 25, admitted November 9th into Mary Ward; married; a night nurse in Stephen Ward; pale looking, weakly woman; had been subject to fits, in which she used to bite her tongue. Skin hot; pain so great in her joints that she cannot move in bed; cheesy exudations on her tonsils. Pulse 120; resp. 48.

November 10th.—Pulse 90; resp. 48; Haust. Efferves. et Quiniæ gr. ij ter die.

11th.—Pericardial rub; great pain in the head, legs, and left arm.

14th.—Resp. 48; sweats much; Julep. Menthæ et Taraxaci ter die; Ext. Opii gr. j in pill, o. n. s.; Brandy ℥ij.

15th.—Pulse 80; throat more sore; no appetite; still in pain.

17th.—Pain in the right shoulder and fingers of the right hand; no brandy to-day.

22nd.—Pulse 80; resp. 30; bowels open; skin cooler; very much better.

30th.—In no pain, but felt weak, but out of bed to-day.

Rheumatic fever, symptoms well marked, second attack.

Mary Ann D—, æt. 20, milliner, admitted May 6th into Mary Ward. She had a slight rheumatic attack when thirteen years old. Present attack commenced fourteen days ago with pains in her face and wrists, then in her ankles. On admission, pains in all her joints; sweats a great deal; perspiration intensely sour; skin hot; tongue furred, yellowish brown; pulse 116; resp. 50; no albumen; heart free from murmur. Ext. Taraxaci, et Julep. Menthæ 6tis horis. Milk diet. Brandy ℥j.

May 9th.—Much the same. Rept. Haust.

12th.—Feels better; not so much pain. Rept. Haust.

15th.—Pulv. Doveri, gr. x hâc nocte.

16th.—Joints still less painful.

18th.—Feels much better; can use her hands, bend her knees, and turn in bed without pain.

19th.—Ol. Ricini ℥ss.

22nd.—Slight pain in her right side, and short of breath.

26th.—Going on well.

30th.—Gets up daily.

June 2nd.—Went out.

Rheumatic fever, symptoms well marked.

Mary Ann S—, æt. 21, single, servant, admitted November 6th, 1861, into Mary Ward; stout built, with sallow skin and dark eyes. Three weeks ago caught cold, her limbs became painful and swollen. On admission left knee and right hand swollen and tender; perspiring very freely; pulse 110 and feeble; resp. 26; slight systolic bruit; urine deposits lithates, no albumen. Ext. Taraxaci ʒj, Ext. Julep. Menthæ 6tis horis; Hyd. c. Cretâ gr. v, statim; Ol. Ricini post. h. s.

8th.—Right hand more painful; to be wrapped in cotton-wool.

9th.—Feels much depressed; right hand not so painful, left tender and swollen.

12th.—Knees most painful. Pulv. Opii gr. j, o. n. Rept. Haust.

16th.—Pulse 86; pain the same.

18th.—Pain and swelling have left the knees, but the shoulder is still painful; sour perspiration; appetite improving; bowels confined. Pulv. Rhei Salinus ℈ij, Ext. Julep. Menthæ statim. Rept. Haust. Taraxaci et Menthæ.

30th.—Left shoulder painful. Adde in singulis dosibus misturæ; Tinct. Calumbæ ʒj. To have an egg and rice pudding.

December 2nd.—Tongue cleaner; appetite good.

5th.—Shoulder less painful; doing well. Pil. Rhei co. gr. v, bis die si opus sit; Quiniæ di Sulphat. gr. ij, bis die 6tis.

8th.—Says she is free from pain. Discharged cured.

A severe case of rheumatic fever; endocarditis.

Elizabeth H—, æt. 12, a servant girl, admitted February 5th, 1864, into Clinical Ward, under the care of Dr. Gull. She had been much exposed to wet and cold. Cannot trace any hereditary tendency. Has had scarlet fever and measles. For three weeks before admission had suffered with headache and pain in her back, and gradual loss of appetite. On February 2nd, that is, three days before admission, she was seized with pain in her limbs, especially in her knees, ankles, and wrists, which were slightly swollen and painful.

February 5th.—On admission, in very great pain, so much so that she dreaded for anyone to touch the bedclothes, or even shake the bed; cheeks flushed; head very hot; perspiration very profuse, and has the peculiar acid smell; wrists and knees seem exceedingly tender to pressure; the superficial veins of the legs are very much distended, and also on the dorsal region of the feet; veins also of the arms enlarged; shoulders and joints, and also the right elbow, swollen. *Heart.*—First sound is prolonged slightly, and there is a slight murmur accompanying it, which is heard most distinctly over the base of the heart. Tongue white, furred, and red at the tip; pulse 102; resp. considerably accelerated; complains of an uneasy sensation in her chest; bowels regular; urine passed freely. Sp. gr. 1022. No albumen or sugar. Ordered—

℞ Ext. Taraxaci ℥ss, Ext. Julep. Menthæ ter die.
Hydr. c. Cretâ gr. iij, statim, et Ol. Ricini, post hor. quat.

8th.—Says that she is worse: the pain in the joints is very intense, and they have been wrapped in cotton-wool; tongue very white, red at the edges. There is a loud murmur over the base of the heart accompanying the systole. Resp. 38. She has pain in the chest of a darting character. Rept. Mist.

10th.—States that she feels better. The pain in the joints not so severe, with the exception of the shoulder, which is worse. Tongue is cleaner; resp. 30; pulse 94; cannot hear a murmur this morning; perspiration in the axilla is alkaline; acid on the left side of the face. Rept. Mist.

12th.—Says she is better; does not sweat so much; joints not so painful; first sound of the heart prolonged, and a slight murmur with that sound; tongue cleaner; appetite pretty good; pulse 84; resp. 30. Rept. Mist.

17th.—Feels better; no pain in the joints; the swelling has disappeared; has no pain in any part of the body; the heart is clear, but the first sound is prolonged; pulse 80, and feeble; resp. 26; appetite good; bowels regular. Rept. Mist.

24th.—Left the hospital cured.

Well-marked case of rheumatic fever, third attack.

Eliza B—, æt. 15, general servant, admitted into Clinical Ward, under Dr. Gull, March 12th. Says that when six years old she had rheumatism, and was ill about three weeks, when she recovered perfectly, and has enjoyed very good health until about last Christmas, when she had pains in her face and a sore throat, and also pains in her wrists, arms, and feet. Has not been exposed to wet or cold, and has worked up to two days ago, when she was compelled to go to bed, and the pain had gradually increased.

On admission, complains of pains in her knees, hands, and ankles, the knees, and thumb of the left hand being swollen. The weight of the bedclothes causes the pain to be intense. Pain worse at night than during the day; skin hot and moist, but does not sweat very much; the perspiration is decidedly acid to test-paper, and there is also the peculiar odour of rheumatism. The face is very much flushed; the tongue coated, white in the centre, and red at the tips and edges; pulse 100, and slightly intermittent; chest normally resonant; respiratory sound normal; first sound of the heart slightly prolonged. No appetite; bowels regular; urine passed freely, contains no albumen, and sp. gr. 1025. Julep. Menthæ et Taraxaci ter die.

14th.—The pain is much more severe to-day. The shoulders and the wrists are swollen, also the hands and knees, and exceedingly painful sweating more freely; perspiration acid; pulse 120; resp. 32. *Heart.*—The first sound is still slightly prolonged, otherwise nothing particular observed. Rept. Mist.

15th.—Pain still very bad. Slept pretty well last night; perspires more than previously; left arm wrapt in cotton-wool; perspiration on the face acid; tongue coated with white fur, red at the tip and edges; pulse 100; resp. 24. Heart in much the same condition. Rept. Mist.

16th.—Says the pain is not so severe, and that she slept better last night. Joints not much swollen; tongue still coated; bowels confined; urine voided scantily; pulse 88; resp. 24. Heart in the same condition. Rept. Mist.

17th.—Pain only in the right hand; perspirations have ceased; tongue a little cleaner; pulse 92; resp. 38. *Heart.*—First sound not so prolonged.

20th.—Pain very slight; tongue clean; appetite very good; pulse 88; resp. 26. Rept. Mist.

21st.—Says that she is in no pain this morning. Tongue clean; swelling almost entirely disappeared; slept very well; pulse 84; resp. 24. *Heart* much the same. She continued from this time to do well.

We would here, moreover, beg to ask further attention to the case of J. G— (p. 477) for the character of the cardiac sounds was very interesting. It has already been more than once mentioned, that the first sound was exceedingly feeble, and, as Dr. Gull remarked, appeared as if it was muffled, or as if it was heard through a layer of cotton-wool. The rhythm of the heart was for a time irregular, and there was doubling of the second sound.

In his excellent work on 'Diseases of the Heart,' Dr. Stokes has recorded two cases (Nos. 13 and 14, pp. 117, 118) of rheumatic endocarditis, in which doubling of the second sound was heard. In Case 13, besides the change in the second sound, the heart's action was, as in J. G—'s case, irregular, its impulse feeble, and the first sound very weak, dull, and muffled.

In the case of a young man lately under the care of Dr. Gull, the peculiar character of the heart's sounds was well shown. The patient had, previous to his admission into Guy's Hospital, come under our notice while attending at the City of London Hospital for Diseases of the Chest, when he complained of pain in his legs, especially in the left knee. He was sweating very freely, his tongue was coated, and he looked ill. The first sound of the heart was so feeble that it was with difficulty heard at all, and this was the case both over the apex and base of the heart, yet there was no indicated area of cardiac dulness; the second sound was doubled in every six or seven beats of the heart; there was no visible impulse below the left nipple, but the impulse was very distinct in the epigastrium. Pulse was very feeble indeed; the respiration was accelerated. On admission into hospital, it was noticed that his power of moving his lower extremities was impaired, especially on the left side, which led to a doubt as to whether the whole condition was not due to some change in the spinal cord. However that might be, it was a good illustration of doubling of the second and exceedingly feeble first sound, and probably the consequence of, as Dr. Gull remarked, some important change in muscular tissue of the heart.

We have recorded the above examples of the disease, some as briefly as possible, others more in detail, with the object of showing the kind of cases in which this plan of treatment was adopted; they were all well marked, and in by far the majority the symptoms were acute. And while some of the cases might be regarded as favorable, others might justly be considered as unfavorable; such as would be likely to put severely to the test, the success of any system of treatment.

We will next proceed to ascertain what the facts observed in these cases tend to show. And the question that we purpose to take first into consideration is the duration of the disease.

The experience in these cases was no exception to what is commonly seen; the number of days that the patient was ill varied very much, as the following observations will show:

George S— (p. 482), ill three days before admission; admitted June 17th; knee-joints painful, much swollen, also left wrist. Treatment began on the 19th. On the 24th, free from and remained free from pain—that is, *on the sixth day of treatment.*

Charles A— (p. 482), also ill three days before admission. Pain and swelling in the left wrist, subsequently in the right knee, afterwards in both hands. Treatment began May 3rd. On the 19th free from pain—that is, on the *sixteenth day.*

Charles H— (p. 483), ill fourteen days before admission. Entered the hospital July 31st. Skin hot, sweating freely, shoulders much swelled; pulse 100. August 3rd, feels much better. 7th.—Free from pain; skin cool—that is, on the *eighth day*.

John H— (p. 484), ill eight days before admission. August 3rd admitted. Pain and swelling of the joints; some perspiration. 5th.—The treatment began. 7th.—Feels better. 10th.—Almost entirely free from pain. 13th.—Completely free from pain; convalescent—that is, on the tenth day of admission, and the *ninth day* from commencement of treatment.

Ellen S—,[1] admitted July 15th, convalescent July 20th—that is, on the *fifth day*.

Elizabeth B— (p. 485), admitted August 2nd, ill fourteen days before admission; joints of the legs swollen and painful. 8th.—Free from pain—that is, on the *seventh day*.

Sarah H—,[1] ill seven days before admission, admitted December 10th. Pain and swelling in the knees and ankles. 15th.—In less pain. 16th.—Pain gone—that is, on the *seventh day*.

William L—,[1] ill ten days before admission. November 25th taken in the hospital, and on the 29th free from pain—that is, on the *fifth day*.

In the above description, it is to be understood, that when it is stated that the pain had abated, the skin was also cool, tongue clean or cleaning, and from that date the patient continued to do well.

These cases we have chosen with the object of showing that the symptoms of rheumatic fever may, unaided by treatment, entirely abate in five, six, seven, or eight days; that the pain in the joints may be severe, swelling marked, skin hot, yet perspiring; tongue furred, and no appetite. All these symptoms may subside and entirely disappear within a week. We have further observed more than once, a patient complaining of pain, accompanied with more or less swelling in one or two joints, with furred and dry tongue; the skin hot, also some perspiration, and on the next morning all these symptoms had disappeared. The sudden and complete cessation of the acute symptoms was observed in these cases, apparently equally as well as is seen in patients who are taking large doses of medicine. The gradual but satisfactory improvement in the symptoms was well seen in a case lately under the care of Dr. Rees in Esther Ward. On the 7th August the skin was hot, yet perspiring very freely; the tongue furred; the knees and wrists were painful, but not much swollen. On the 9th August the skin was cool, tongue clean, pain almost entirely gone, and the patient expressed herself as feeling much better; yet that patient had not had a dose of medicine except Aqua Menthæ ter die. Milk diet. Another case, also in Esther Ward, and under the care of Dr. Rees, showed well the steady improvement in the symptoms, as seen below. August 14th, on admission the patient complained of great pain in the hips and knees, and the latter joints were swollen; skin hot, with the rheumatic odour, and perspiring very much. Ordered Aqua Menthæ ter die. Milk diet. On the 16th she stated that she was not in so much pain, but the joints of the right hand were swollen and painful; the tongue thickly coated with yellow fur, and red at the sides; perspiring very freely, with the rheumatic odour strongly marked; pulse full and regular; little or no appetite. 18th.—Still in pain, otherwise much the same. 22nd.—Tongue still thickly coated; sweating freely; bowels had not been open for three days; joints still very painful, and especially the wrists; the urine high coloured, and a deposit of lithates; sp. gr. of the urine 1030. On the 23rd in no pain; appetite improving. 24th.—Remains still free from pain; tongue cleaning; appetite "getting better;" skin cool, and not perspiring; the saliva has become alkaline, and perspiration is either neutral or faintly alkaline, certainly not acid. 27th.—Tongue much cleaner; appetite very much better; not in any pain, but perspiring freely; pulse 73; resp. 26; urine about the average quantity, acid reaction. September 1st.—Urine sp. gr.

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[1] The records of these three cases do not appear to have been included in this paper.—Ed.

1025, faintly acid; saliva alkaline; in no pain. 2nd.—States that she feels very well, free from pain; urine alkaline. This patient also had no medicine, excepting Aqua Menthæ ℥j ter die. Milk diet. We have not mentioned the condition of the heart in this case, and it is probably sufficient to say that a systolic bruit was heard over the apex of the heart, and also in the axilla; it was observed on the day of admission, and was probably due to a previous attack of rheumatic fever, of which the patient gave an account.

This was a typical instance of rhematic fever; the symptoms were markedly acute. The progress of this case will probably be regarded as favorable, and such as would have done credit to any system of treatment. It was especially satisfactory to observe in this case, that although the bowels had not been moved for three days, the sp. gr. of the urine was as high as 1030, and the tongue thickly coated, nevertheless, two days afterwards, the bowels were relieved without the use of medicine, urine of lower specific gravity, the tongue and other symptoms gradually improving.

When we refer to those cases in which not only were the symptoms acute, but there was undoubted evidence of pericarditis, or of endocarditis, we observe that the duration of the disease was increased, as may be seen by the following abstracts:—

The case of George E— (p. 476), admitted June 5th, aged 24, was a good example of this disease; the symptoms were acute; there were pain and swelling of the joints, accompanied by other attendants of the fever, pericarditis and endocarditis. A to-and-fro murmur was distinctly heard over the region of the heart, but no evidence of any great effusion into the pericardium. The patient complained of pain over the region of the apex of the heart, which was especially felt during forced inspiration. Also a soft murmur was heard over the base of the heart. No especial treatment was adopted for either the arthritic symptoms or the inflammation of the pericardium. The friction sound continued to be heard during thirteen days, it then disappeared, and in its place a soft systolic bruit was heard over the apex, which remained even when the patient left the hospital. This patient was thirty-two days in the hospital, and at the end of that time went out apparently well, but with indications of a damaged mitral. The treatment began on the 6th of June. On the fourth day, that is, on the 9th of June, his joints were free from pain; on the seventh day of treatment, that is the 12th of June, his skin was cool, and all the pain and swelling of the joints had gone. On the 25th of June, that is, the twentieth day of treatment, he was out of bed, and completely convalescent, and continued from that time to do well. In this case there was, on the day of admission, undoubted evidence of cardiac inflammation, therefore any endocardial mischief that was left could not fairly be attributed to the treatment.

The case of John S— (p. 478), aged 22, was another good example of rheumatic fever with inflammation of the pericardium and with the addition of albuminous urine. It appeared to be his third attack; and during the first he stated he had been laid up two months. The last attack began, as is not uncommonly seen, by face-ache. On the day of admission the signs of pericarditis were well marked, also great pain in all his limbs, but no swelling. Pulse 132; respiration 40. On the day after admission his countenance was anxious: he complained of great pain in the chest. Pulse 120; resp. 50. He was ordered Julep. Menthæ c. Tarax. ter die.

Having had a grain of opium on the night of the 31st of October, it was found on the morning of the fourth day after admission into the hospital that he had slept well, and that there was less pain in the chest, but the knee was painful, and the mean specific gravity 1026, and albuminous. Pulse 120; resp. 48. On the seventh day after admission he could move his hand and arms much better; and the pericardial rub could scarcely be heard. Eighth day an eruption of sudamina was noticed. On the thirteenth day he seemed much better. On the eighteenth the rheumatic symptoms seemed to have disappeared. The pulse had fallen from 132 to 90, and the respiration from 50 to 22 a minute. He continued daily to improve, and went out of the hospital on the 25th of November—that is, on the twenty-eighth day after admission.

We would next refer to the case of Elizabeth H— (p. 487), aged 12, who was admitted into the clinical ward 5th February, 1864, under the care of Dr. Gull. It is stated that she had been ailing three weeks, but had only suffered pain in the joints for three days before admission. February 5th, the symptoms were acute; the pain was so great that it is stated she dreaded the bedclothes to be moved. Her cheeks were flushed, and her skin hot, but perspiring profusely, with the well-known rheumatic odour; the joints were swollen; a prolonged first sound heard over the base of the heart; her pulse was 102; urine sp. gr. 1022. No albumen or sugar. She was ordered the Julep. Menthæ ℥j ter die, and Hydr. c. Cretâ gr. iiss stat., Ol. Ricini post hor. quat. On the third day of her residence in the hospital the pain got much worse. A prolonged first sound was heard over the base of the heart; resp. 38. On the fifth day she felt much better. Resp. 30; pulse 94. The perspiration was neutral, or perhaps slightly alkaline. On the seventh day she stated that she felt better; she did not sweat so much. Pain was not so severe; tongue cleaning; her appetite was pretty good. Heart, first sound prolonged; the pulse had fallen from 102 to 84. On the *twelfth day* she was free from pain and swelling. Pulse 80; resp. 26. Appetite good; first sound of the heart still prolonged. She continued after this to do well, and left the hospital February 24th, apparently cured. In this girl the rheumatic symptoms were markedly acute, the pain was very great, and yet the pain was better on the seventh day, and the appetite pretty good. On the thirteenth day free from pain. Pulse 80; resp. 26; appetite good; continued to do well, and she left the hospital on the twentieth day.

To recall the results seen in these cases: one patient was totally free from pain on the fifth day after admission; another on the thirteenth day after admission; a third on the thirteenth day also. In the first case the duration in the hospital was thirty-two days, in the second twenty-eight days, in the third twenty days.

In the following cases there was evidence of cardiac disease:

Thomas F— (p. 480), admitted March 2nd. Physical signs of aortic regurgitation, the injury to the valves believed to be due to a previous attack of rheumatic fever. On the fourth day of admission he was free from pain. On the eighth day the pain returned, although slightly. On the eleventh day better again. On the thirteenth day out of bed. On the nineteenth day again in pain, wrists swollen. Pulse 140. On the twenty-third day in less pain. *On the twenty-sixth day again better. On the twenty-ninth day* went out of the hospital better, although evidently not quite well.

The next is a very instructive case.

Jane C— (p. 485), admitted November 18th, had evidence of pleuritic effusion on the right side, of acute pleurisy without effusion on the left side, and feeble systolic bruit heard over the heart. Yet on December 3rd, that is, the sixteenth day after admission, she was free from pain. On December 6th, that is, on the nineteenth day after admission, she was out of bed; and on December 13th, *on the twenty-fifth day* after her admission, she had quite recovered, and went out of the hospital.

Emily W— (p. 486), admitted November 9th. On the 11th of November a pericardial rub was detected. On the 30th so much improved that she was allowed to get up—that is, on the twenty-second day of admission.

We venture to make one more reference to the case of John G— (p. 477), admitted June 6th; it was a very instructive and severe one. The heart was apparently involved when admitted. At no period of his illness were there any physical signs of pericarditis, but the first sound of the heart was exceedingly feeble, as if it was muffled, and that, taken with the subsequent irregular rhythm and doubling of the second sound, led Dr. Gull to consider that the muscular tissue of the heart was much more involved than the sero-fibrous tissue. The external symptoms were acute. The joints were very painful and swollen, and there was profuse sour perspiration. He appeared very ill, and expressed himself as feeling so. On June 25th, that is, on the twentieth day, the heart's action was still irregular, but the pains had entirely gone. On July 1st, that is, on the twenty-fifth day, he was entirely free from pain, and in other respects much better—so much so that Dr. Gull ordered him decoction of cinchona, with sesquicarbonate of soda three times a day. On July 7th, that is, the thirty-first day, he was again in great pain all over his body. Skin again hot and perspiring, and he complained of so much pain in his left chest that Dr. Gull ordered a blister to be applied over the region of the heart, which he stated gave him great relief. On July 14th, the thirty-eighth day, he was a second time free from pain, and continued afterwards to do well.

Thus, on referring back, it may be seen that the average duration of the acute symptoms in the first seven cases, in which there was no evidence of the heart being involved, the abstracts of which have been just given, was 8·5 days; while in the last six cases, in which the heart was evidently affected, the average duration was 23·6 days. The recorded experience of other hospital physicians confirms what is here shown—that cases in which there is not, on admission into the hospital, evidence of much heart affection, tend to get well in from six to fourteen days, that is within twenty days; cases, however, in which there are very early indications of severe heart affection, tend to last over twenty days.

In the cases with little or no cardiac disease, the shortest duration of the acute symptoms was six days, the longest sixteen days. Where the cardiac disease was great, the shortest duration of acute symptoms was seven days, the longest thirty-eight days. Many of the cases tend to confirm the experience, as late years have shown, that acute rheumatic pericarditis does not, as regards the recovery of the patient and the successful termination of the disease within a reasonable time, require any special treatment; and more than that, it shows a patient may have rheumatic inflammation of both pleuræ, and effusion into the right; and that the heart also may be involved; yet in thirteen days, all the acute changes may subside, and the patient be convalescent and out of bed on the twenty-fourth day, and the only treatment adopted be a grain of extract of opium every night, and mint julep with extract of dandelion three times a day.

Such facts also tend to teach that acute rheumatic pericarditis may subside, without any treatment, except rest in bed, and careful diet, in fourteen days; that rheumatic pericarditis complicated with albuminous urine, may, without medicine, except mint julep and a grain of opium, for one night only, have so far recovered as scarcely to be detected on the seventh day of treatment, and the patient afterwards continue steadily to improve and able to go out of the hospital within a month.

For some further observations on this point see Dr. G. Gairdner's instructive 'Essay on Pericarditis.'

We next propose to inquire if there is any evidence to show that this kind of treatment rendered the heart more than ordinarily liable to rheumatic inflammation.

We would here beg to remark that it is Dr. Gull's impression that the cases, so treated, were not more liable to suffer with heart complication. Yet the difficulty at arriving at any very exact conclusion was, as it must always be, as long as the investigation is confined to hospital patients, very great. The chief obstacle is found in the fact that so very few patients come into the hospital who have not already some signs of cardiac affection, or who have not previously suffered with rheumatic fever; and although there might be no bruit to indicate that the previous attack had injured the heart, yet it will probably be allowed that the absence of murmur is no proof that the endocardium or pericardium has not been damaged, thereby rendering the heart particularly liable to be attacked by any subsequent rheumatic inflammation.

The condition of the heart in these cases was as follows:

Evidence of pericarditis . . . . . . in	6
A bruit mostly heard at the apex . . .	17
Rhythm of the heart irregular, but no bruit .	2
First sound prolonged, but no actual bruit .	2
Suspected myocarditis	1
Not stated	2
No abnormal sounds	11
Total .	41

In nearly all, if not in all the above cases, the signs of cardiac disease were detected either on the day of or within two or three days after admission. This, however, depended upon how long after admission Dr. Gull saw the patient, for as Wednesday is the taking-in day at the hospital, and the Friday afterwards Dr. Gull's next day of attendance, two days would elapse. That is the reason why in some cases the heart is not mentioned until the second or third day after admission.

And we would here observe that it is very difficult in some cases, as will probably be allowed, to say whether the heart has or has not been seriously injured.

When the symptoms are very acute, and the changes in the pericardium or endocardium acute also, the physical signs are usually well marked; but when the symptoms are subacute and somewhat chronic, the changes are slower and more difficult to detect. It is generally easy to detect the friction sound in acute pleurisy, next to impossible to diagnose, by physical signs, those slow changes which give rise to greatly thickened pleuræ.

There is evidence tending to show, that although the rheumatic inflammation may have seriously injured the mitral orifice, yet a certain time must elapse before that is evident by physical signs.

Further, that the muscular tissues of the heart may have undergone degenerative changes, the consequence of the rheumatic inflammation, and subsequent post-mortem examination show the pericardium to be healthy, the left ventricle dilated and attenuated; and in such cases there may or may not be a bruit.

It will be probably considered that, where the muscular tissue has been damaged, some time must elapse before we have all the signs of dilatation of the left ventricle, of the mitral orifice, and subsequent changes.

A very good example of this condition may be seen in the post-mortem records of Guy's Hospital for the year 1860; No. of record 180.

Samuel B—, aged 29, a patient of Dr. Habershon's, had had a severe attack of rheumatic fever two years before. For six weeks before admission into the hospital he had suffered with severe pain "in the heart," pain in his stomach, and shortness of breath. On admission a loud systolic bruit was detected beneath the left nipple, and faintly heard in the axilla; the abdomen was tender; the liver enlarged; œdema of the feet and legs; no albumen in the urine. Autopsy, by Dr. Wilks, showed the condition known as "heart lungs," with some lobular pneumonia. Heart enlarged in all its cavities; left ventricle dilated; mitral orifice very large, no doubt admitting regurgitation, but the valve itself healthy; pericardium healthy; kidneys healthy; nutmeg liver.

There is a patient now under us at the Victoria Park Hospital, who complains of shortness of breath on exertion, and "weakness." The apex of the heart is three inches below and directly under the nipple; no visible impulse. The first sound extremely feeble; a sharp clicking second sound, but no bruit. He stated that two years ago he was laid up twelve weeks with rheumatic fever. There are no physical signs of emphysema or of any organic change to push down the apex of the heart.

Some years ago a boy about twelve years old was under the care of Dr. Gull for, and died of, rheumatic fever in the hospital.

The post-mortem examination showed the heart had undergone extreme fatty change; the valves and pericardium healthy.

It would appear that in order to know whether the heart has been injured during the rheumatic fever or not, the patient should be examined six weeks or two months after the rheumatic attack.

Some very interesting facts were seen, in relation with albuminous urine and rheumatic fever, which we must defer until the next number of the 'Reports,'[1] when we hope to bring forward still further evidence and records of more cases treated on the same plan; more especially as Dr. Rees proposes to treat a given number of cases with mint water, and an equal number with alkalies, lemon-juice, &c.

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[1] "A Second Report of Cases of Acute Rheumatism treated in the Wards of Guy's Hospital, with Remarks on the Natural History of the Disease," by Henry Sutton, M.B., 'Guy's Hospital Reports,' Series III, vol. xii, p. 509. The majority of these cases were under the care of Sir William Gull, and they were treated for the most part by mint water alone.

On referring to the above cases it will be seen that there did not appear to be any great disposition to relapse.

On looking back, and taking into consideration what has been already stated, it would appear that cases of rheumatic fever in which the symptoms are acute, and in which there is no, or very slight cardiac affection, tend to get well in seven to fourteen days; or probably, we may safely say, under twenty days. The majority get well under a fortnight, but it would appear that in order that such may be the case it is necessary that the symptoms should be prominent and acute, that the patient should be of a tolerably good constitution, and that his tissues should not have been damaged by previous disease. When, however, we refer to those cases in which the symptoms were subacute, or in which the patient's constitution had been weakened, we cannot but see that there was a marked difference, and the duration of the disease was much longer. In order, however, more clearly to demonstrate this, we would ask attention to the following abstracts:

Henry J— (p. 480), aged 26, described as a pale, weakly-looking man, who had been ill fourteen days. When admitted complained of pain in his hands. He perspired freely during the night. Besides the mint julep he was ordered half a grain of opium every night and morning, with two ounces of brandy a day. On the fifth he felt better; pulse 80; but a systolic bruit was heard over the base of the heart, and increased dulness on percussion over the cardiac region. On the seventh day he was in a good deal of pain, and passed a very bad night. The treatment was continued, but in addition he was ordered to have half an ounce of castor oil. The signs of pericardial effusion remained, but no friction murmur was heard. On the thirteenth day he was free from pain, but on the nineteenth day apparently worse than ever. Pulse 120; pain and perspiration had returned. On the twentieth day he was ordered Haust. Quiniæ ter die. The next three or four days he improved much, and his pulse fell to 76. After that he gradually got well, and left the hospital November 26th, having been admitted October 15th—that is, forty-three days in the hospital.

Another case of a similar kind as the last was that of Elizabeth D—, aged 18, in Esther Ward, under the care of Dr. Rees. She was a girl with regular features, face very pale, and stated she had always been delicate.

August 3rd.—On admission into the hospital she complained of pain in her left wrist, but it was not much swollen; also of pains in her knees. Her skin was not hot, but she remarked "when asleep, I sweat a good deal." Her tongue was clean, but rather red; pulse was not quick, nor the respiration either. Percussion over the cardiac region showed resonance somewhat diminished, but no actual dulness, as high as the second rib. The heart's visible impulse was diffused and very distinctly seen. Over the third left costal cartilage and third left interspace, also between the second and third rib, a murmur was heard, such as is usually likened to the unfolding of parchment. Besides this a more superficial crackling sound, as if the pleura over the pericardium was involved; the latter was more distinctly heard during inspiration. A well-marked systolic bruit was heard on listening over the carotids. She complained of soreness and tenderness across the chest. August 5th, that was the second day in the hospital, the physical signs were the same —no perspiration, no particular heat of skin—the rheumatic odour faintly marked; wrists painful. On the fourth day in the hospital, that was August 7th, the skin was hot, perspiring very freely; tongue furred; pain in the knees and wrists, but no swelling, and no pericardial murmur could be detected; but the first sound of the heart over the base was much prolonged. On the sixth day after admission the skin was again cool, and the pain entirely gone, except a little in the ankle; and she stated that she felt much better. The first sound at the base still prolonged. August 11th, that is on the eighth day after admission, the shoulders were very painful and stiff, but the tongue was clean, and she was perspiring freely; pulse not particularly quick, but full and bounding; face pale. August 12th, that was the ninth day, pain in her left wrist; skin neither hot nor perspiring; tongue slightly furred at the base; enjoyed her dinner.

There was diminished resonance over the cardiac region, but no actual dulness as high up as the third rib. First sound at the base prolonged; visible impulse diffused and very distinctly seen. On the tenth day her breathing was quick, and she said that she could not lie on her left side, as immediately she did so it gave her pain right through her chest, pointing to the left side. No cough; the right wrist painful, the pain had left the ankle; she did not perspire much, and her skin had not any very distinct rheumatic odour. Heart's sounds the same. On the 16th August, the thirteenth day, ankles again painful, not swollen; tongue clean and moist; pulse rather quick and feeble. She remained much the same until 18th August, that was the fifteenth day after admission, when again she was free from pain; appetite good; skin cool; tongue clean. The first sound at the base less prolonged. She got out of bed without leave, and remained up three hours, and next morning the pain had returned into the ankles. She perspired when asleep, but she did not lose her appetite. August 22nd, the twenty-first day in the hospital, her pulse was 80, feeble; tongue clean; resp. 28; no perspirations, except when asleep; no rheumatic odour; free from pain, except an occasional shooting pain in the right hand; appetite good; sleeps well, and says that she feels a great deal better. The first sound of the heart is prolonged the same as it was ten days ago. The urine of sp. gr. 1012, pale-looking, highly acid, and when boiled with the addition of nitric acid showed a trace of albumen. August 25th, the twenty-second day, the symptoms were much the same; she thought herself well, and asked to be allowed to get up. Saliva alkaline, perspiration also. Heart sound at the base still prolonged. 26th.—Urine alkaline. 27th.—All the symptoms had subsided. September 1st, the thirtieth day, she was out of bed, but complained of pain in the ankles. Saliva acid; tongue moist and clean; skin smells rather sour again. 2nd.—Urine alkaline; saliva alkaline also; pain in the right knee; heart much the same. Up to this date she had taken no medicine of any kind excepting mint water; no stimulants; milk diet the first fortnight, and then middle diet; but as she had lingered on, one day better and the next day worse, she was ordered three ounces of brandy a day, mint water three times a day, and middle diet and to be up every day.

She, however, did not improve much; the pain varied, and more than once she was compelled to remain in bed a day or two to get rid of the pain in the joints, mostly the ankles, and the joints of the fingers. All this time the tongue was quite clean, and the appetite was very good; the saliva alkaline; urine varied, at one time acid, at another alkaline. The prolonged first sound became more and more marked, and at present—for she is still in the hospital—it would be considered to be a decided murmur; and although she is now free from pain, and has been the last three or four days, yet for forty-four days she was more or less in pain, with other evidence of the rheumatic state, and indications of cardiac disease.

The average duration of the last two cases was respectively forty to forty-four days.

The latter class of case will be, we have no doubt, at once recognised as a not uncommon variety of rheumatic fever, and we have introduced it here on purpose to contrast it with the preceding cases, and to show how far the plan of treatment that is applicable to one is also applicable to the other, and, above all, to point out how far each, unaided, tends to get well.

It would appear, from the above evidence, that those cases in which the symptoms are acute tend to get well much sooner than those in which the symptoms are subacute, and this agrees with what has been stated by Chomel in his 'Inaugural Dissertation:'—"Dans le rhumatisme aigu elle s'etend rarement au delà du deuxième ou troisième septenaire, quand il est intense, et du sixième quand il est léger." (See Bouillaud, 'Rhumatisme articulaire,' p. 292.)

Here we would mention that we have heard Dr. Gull several times observe that to have prominent and easily detectable symptoms is a good sign, for when the system is very low, an organ may be very much diseased without being able to tell us so. With respect to the rheumatic fever, the hot skin, the copious acid perspiration, the high colour, and the high specific gravity and acidity of the urine, the sensibility to, and the acknowledgment of pain are good signs, for in some of the very worst cases of acute rheumatism the perspiration is alkaline ; the arthritis slightly marked ; the skin does not perspire freely, nor is there much or any pain.

The experience gained in these cases would appear to confirm Dr. Gull's remark.

It may also be noticed that in those cases in which the symptoms were acute, the patients had for the most part enjoyed good health previous to the rheumatic fever ; and when we further call to mind that general experience has long taught that acute sthenic symptoms occur for the most part in the healthy and robust, asthenic symptoms in the weak and delicate, we are led to consider that the acute cases recover well, not because they are severe, but because their severity indicates that the system has sufficient power not only to tell us of its sufferings, but further, that it is capable of making a great effort to restore its tissues to a more healthy state of nutrition, and to remove the unhealthy matter by active elimination.

Here we would observe that the clinical experience of other physicians has shown that those cases tend to do the best in which the skin is rather hot, yet perspiring with the rheumatic odour, and the pain distinctly marked (see Dr. Fuller's well-known work on 'Acute Rheumatism')—thus agreeing with what Dr. Gull's cases tend to teach.

Before, however, we conclude that acute sthenic symptoms are good indications, we would wish to inquire how far such a conclusion can be supported by what is seen in other acute diseases.

At the time that diseases were regarded as entities, when acute inflammation was looked upon as a sudden and rapid organic change excited by accidental circumstances in some part of a healthy body, it would have been impossible not to agree with the acknowledged opinion that severe and well-marked symptoms were not only a measure of the intensity of the inflammation, but that they were also indicative of the danger to which the patient was exposed. The careful study, however, of morbid anatomy has shown that acute inflammation is not simply active and rapid cell-formation in healthy tissue, but rather acute change supervening upon chronic mal-nutrition or degeneration of tissue (see Dr.

Wilks's remarks in the 'Guy's Hospital Reports,' Third Series, vol. iv).

And further, in confirmation of this observation, clinical experience has taught that the more the tissues have degenerated, the less evidence they appear capable of giving to our senses that they are inflamed; that the earlier the age, and the more vigorous the constitution, the more marked the symptoms of inflammation; whilst the older the life and the more broken down the constitution, the less openly do acute changes in the tissues betray themselves.

The history of acute pneumonia illustrates this statement. A man who has lived regularly, and who is of a tolerably good constitution, is suddenly attacked, in the midst of apparent health, with inflammation of the lungs. His skin becomes burning hot, his breathing rapid, his expectoration rusty. The physical signs are very well marked, and show that the lower half of one lung is consolidated. Such a man, experience has shown, generally does well, for not only does the disease run gradually to resolution, but, more than that, after resolution has taken place, the new matter is apparently completely removed, and no secondary change follows; no new chronic cell-formation has been brought into existence. Whereas, if inflammation attacks an old man, or one who has drunk hard—whose tissues have degenerated, the patient complains much less, the symptoms are less evident, the physical signs are less, distinctly marked. The acute change may apparently subside, but from that day the patient is noticed never to be well. After a few months or a year or two he dies, and the autopsy shows that a new cell-formation, akin to a growth, has steadily gone on apparently ever since, and in the same seat as the previous inflammation.

Similar experience is seen in cases of acute pleurisy, when we contrast the symptoms seen in a vigorous young man, coming on after rowing hard, or some other such determining cause, with those seen in a man who is suffering with granular degeneration of the kidneys, emphysematous lungs, and other degenerative changes.

If, however, we compare the natural history of acute rheumatism with that of gout, we see a still closer analogy.

Observation has long taught, beyond all question of doubt, that if a man be highly gouty and has an attack of gouty inflammation of the toe, it is followed by great relief to the system. The agony is great, and the inflammation appears great, yet after a time it subsides and the patient expresses himself better than before the attack. It is supposed that the severity of the attack corresponds with the amount of gouty poison in the blood, and the inflammation is a "depurating process." Certain it is that when the tissues of gouty men have degenerated, the attacks of gout are no longer marked with the same severity. Symptoms feebly marked appear more frequently, and are looked upon as evidence of "mild attacks;" the intervals between the attacks are shortened, and the patient is no longer sensible of much relief. To finish the comparison, it is further well known that a patient, subject to gout, may be very low, depressed, feeling and appearing weak, occasionally seem to be getting somewhat quickly anæmic; his urine is examined, and found not to contain any albumen, but neutral, or occasionally found to be alkaline. The same patient has an attack of gout—the urine becomes acid, high-coloured, of high specific gravity, loaded with lithates, and he gets marked relief.

If, therefore, what has been last stated be correct, we are led to regard acute sthenic symptoms as indicative of a highly disordered state of the system, but also evidence of a certain amount of power in the system.

Other illustrations might be brought forward, but we may probably appeal to general experience to show that the very acute diseases that physicians formerly regarded as sthenic, and requiring active treatment, are now found to be those that tend to get well without the aid of medicine.

Let us now refer back to the case of Elizabeth D—, under the care of Dr. Rees, where, as we stated, there was no perspiration except when asleep, and, during a greater part of the attack, the skin was cool, the tongue was moist and clean, and she had very slight pain, but there was evidence of pericardial and probably of endocardial disease, and the latter was becoming more and more unmistakable every day. The urine was generally of pale colour and of low specific gravity. One day the perspiration was neutral or alkaline, and little or no pain; another day the skin rather hot, perspiring rather freely; the perspiration had an acid reaction, the saliva also, and the pain and tenderness in joints acute. In fact, when the pain was acute, and the skin hot, the perspiration was acid; when the skin was cool, and little or no pain, the perspiration was neutral or alkaline. So the case continued for over six weeks. The frequently repeated relapses showed clearly that the rheumatic condition remained; and although the external rheumatic symptoms were altered, the cardiac mischief apparently progressed. In fact, the symptoms disappeared, but the disease did not. The patient was a weak, delicate-looking girl, and her system did not appear to have sufficient power to cast off the unhealthy matter. Every now and then it seemed to make an effort, but had not the strength to continue. The disease tended to become chronic, and, as a consequence, to become insidious.

This case would appear to show that we may have a rheumatic condition of the system without any rheumatic symptoms. That a person may have the rheumatic fever without the rheumatism, the fever without the arthritis, we are all well aware some of the most distinguished minds of our profession have believed and affirmed.

We are every now and then coming across cases in which we learn that a patient does not feel well, thinks she loses flesh, and her friends notice that she is getting pale, she complains of occasional slight pain, and is apt to perspire freely; no physical signs to indicate chest, and no evidence of other organic disease. A week afterwards we learn that while going from the hospital she caught cold, and the day afterwards the pain fixed itself in a joint; in a few days more there is undoubted evidence of rheumatic fever. She recovers, and is apparently better than before her illness.

A young girl was in the Clinical Ward last year, under the care of Dr. Gull, for paralysis of the face. She had slight wandering pains, and occasional perspiration, which gave an acid reaction to test-paper. She remained about the ward for three weeks, and at the end of that time it was quite clear that she was suffering with rheumatic fever and rheumatic endocarditis.

A patient now under our care, at the City of London Hospital for Diseases of the Chest, complains of shortness of breath, cough, and of other symptoms. A systolic murmur can be heard over the apex of the heart. On inquiry, she stated that she had never to her knowledge had either scarlet or rheumatic fever; but on further inquiry she stated that about two years ago she attended the London Hospital as an out-patient, when she felt very weak, and that she then complained of pains flying about her. She, however, got better, but the last few weeks the pains had been gradually getting "bad" again.

A young lady was suffering from sore throat; she was very subject to enlarged tonsils. Some yellow cheesy-looking substance was noticed on the tonsils. She appeared depressed, and was sweating very freely, but no acid odour. She was kept in bed for three days, then appearing much better, she was allowed to get up. She seemed to improve, but her friends remarked that they could not tell why she did not gain her strength as fast as she had done after her previous attacks of sore throat. One day, about a month after she had had this throat affection, she complained of pain in the right knee. Her skin was cool, but when asleep she perspired, but not very much; no rheumatic odour; tongue clean; her pulse not particularly quick. For precaution's sake she was kept in bed, and ordered half-drachm doses of bicarbonate of potash with lemon juice, three times a day, and milk diet. There was pain, first in one knee, then in the other, also in the ankles, but no swelling; so she continued for eight or ten days. She complained very bitterly because she was kept in bed, for, as she expressed it, "I don't feel ill." About the end of that time the pain became more severe; the knees and wrists swelled, and the skin became hot, but still not much perspiration; tongue rather furred. A harsh, grating, to-and-fro murmur was heard over the heart. This case got gradually worse and worse, the dyspnœa became so great, or rather the breathing became so accelerated, that it was most distressing to see her. She recovered in about seven weeks, but still a loud systolic murmur is heard over the apex and over the angle of the left scapula.

We may probably appeal to observation to show that it is not very uncommon to see a young person, who appears as if she is threatened with phthisis, seized with rheumatic fever, and after completely recovering from the fever, appears to be in much better health than before the attack. When speaking of experience like this, Dr. Gull remarked,—rheumatic fever sometimes appears as if it saved a person from phthisis; and it has certainly seemed so to us.

Is there not evidence in all this, to lead us to think that there is a rheumatic condition of the system, that we may not be able at present to recognise by symptoms, and yet a slight exposure to cold, or any such determining cause, may be the means of exciting acute changes upon chronic rheumatic conditions? It is in the many different ways the same morbid state may manifest itself, that we find the difficulty in collecting statistical evidence about any given disease. We have a group of symptoms which indicate certain morbid conditions, and we assign a name; but when the diseased condition is there, and not the symptoms, or not the same symptoms, we are at a loss.

In relation with the rheumatic state, is the very important question, when we have lost all the symptoms of rheumatic fever,—has the patient lost the rheumatic condition of the system? The case of Dr. Rees would lead us to think not. Moreover, we would further call to mind, that although all the acute symptoms may have ceased, yet the evidence of endocardial change may go on day by day increasing, till at the end of six or eight weeks, that which was a prolonged first sound at the apex becomes an undoubted bruit. There was, in the clinical ward under Dr. Wilks, a case of rheumatic fever, with severe acute symptoms. Blisters were applied to the joints; the patient got very speedy relief, and in a few days was out of bed. The heart had been carefully watched day by day, and while in bed no abnormal sounds were detected. After she had been up some days, the first sound over the apex of the heart was noticed to be much prolonged, and each day afterwards it was more easily detected; when she went out of the hospital there was a marked systolic bruit at the apex. We do not imply that the blisters had anything to do with the after change, for the same fact is observed after other forms of treatment.

Would not the above observations lead us to think that under treatment we may perhaps suppress the symptoms without curing the disease? The very cases in which we should fear that we might do so are the subacute, which tend to become chronic and insidious. When the constitution is vigorous, and the symptoms acute, it would, perhaps, be much more difficult to do so. When we have acute symptoms, however, the disease tends to get well; the acid secretions tell us how great is the effort that the body is making: it may succeed, and the patient be well in a few days; it may not have strength to finish the struggle, and the continuance of the symptoms day by day, and week by week, would probably indicate that the system is becoming exhausted, and wants assistance.

We would here finish these observations by remarking that we do not wish it to be understood that Dr. Gull considers that we ought to leave rheumatic fever to run its own course, but rather that the profuse acid perspiration, the acid saliva, highly acid urine of high specific gravity, and the other acute symptoms, are to be looked upon as favorable signs,—that although they may be evidence of a highly rheumatic state of the system, there is also evidence, as these cases show, that the disease tends unaided to get well. Moreover we would beg to mention that the natural history of inflammation has shown that the acute changes may subside, but the chronic changes still go on, rendering a vital organ less and less competent to perform those functions which are essential to our existence. The chronic condition may continue until some accidental circumstance again sets up acute changes, and completely exhausts the only power remaining in an organ essential to life. So it appears to be with rheumatic fever. A patient has an attack of that disease, recovers, and appears well for a time; some comparatively slight cause brings about another attack; this may be again and again repeated; each attack damages the heart still more, until the muscular tissue has so degenerated that it is unable to perform its functions properly. The lungs in consequence become en-gorged. At last the patient dies, and the immediate cause of death is seen in the condition of the so-called "heart lung."

The fact that comparatively slight causes, such as would have had at one time no effect, are at another time capable of exciting the disease, would seem to show that there is a chronic condition, favouring the action of such causes.

We would here, in ending these remarks, venture to quote Dr. Latham's words :—"There is a lesson which we are apt to learn slowly, but all of us learn at last: it is this, that while present pain and present peril call loudly for relief and rescue, still, in relieving and rescuing, the ultimate well-being of the patient must not be disregarded altogether."

A perusal of the above cases tends to show that the best treatment for rheumatic fever has still to be determined, and will also convince the reader (we think) that it is absolutely necessary to understand the natural progress of the disease before any conclusion can be arrived at concerning the operation of remedies. The cases show that too much importance has been attached to the use of medicines, especially in those acute cases where the tendency to a natural cure is the greatest.

REMARKS

ON THE

NATURAL HISTORY OF RHEUMATIC FEVER.[1]

In this paper we are desirious of bringing under the notice of the profession the particulars of a few more cases of rheumatic fever which have been treated by mint water, or, in other words, by absolute rest and regulated diet, unaided by medicine.

In our previous reports on this subject (recorded in the 'Guy's Hospital Reports') we have endeavoured to show what is the natural course of rheumatic fever.

We have, moreover, shown what is the class of case that tends to do well, to recover more quickly than any other class; and have further pointed out what class of case does not tend to recover quickly, but becomes protracted and very liable to relapse.

We are now desirous of pointing out what appears to be the natural course of rheumatic fever with reference to the heart; to show in what proportion of cases the heart has become diseased when the patients were treated by mint water; and to consider if there be any evidence to prove that the heart is more frequently involved where cases are treated by mint water, or where treated by alkalies, lemon-juice, or by the application of blisters to the joints.

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[1] By William W. Gull, M.D., D.C.L., F.R.S., and Henry G. Sutton, M.B. Reprinted from 'Medico-Chirurgical Transactions,' vol. lii, 1869, p. 43.

This report is based on twenty-five cases of rheumatic fever, all of which have been carefully watched by one of us.

Four of these cases were under the care of Dr. George Owen Rees in Guy's Hospital (*vide* 'Guy's Hospital Reports,' vol. xi, third series, 1865); eight of the cases occurred in the same hospital under the care of Drs. Barlow, Rees, and Gull (*vide* 'Guy's Hospital Reports,' vol. xii, third series, 1866); and the remaining thirteen cases have occurred in the London Hospital. The thirteen patients were admitted into the London Hospital under the care of Dr. Herbert Davies, Dr. Andrew Clarke, and Dr. Ramskill. They were, however, subsequently transferred to the care of Dr. H. G. Sutton, who here begs to acknowledge the kindness and liberality of his colleagues, and to mention that Drs. Davies, Clarke, and Ramskill have at all times most generously allowed him to make use of their cases.

Twelve of these twenty-five cases have been already recorded in the 'Guy's Hospital Reports;'[1] it is therefore only necessary to give the clinical particulars of the remaining thirteen cases.

Of these 25 cases, 18 were females and 7 were males.

The ages of these patients were as follows:—The youngest was twelve years old and the oldest was twenty-five years. The average age was nineteen years. No objection could therefore be raised against the cases on account of age, for experience has shown that persons under twenty suffering from rheumatic fever are very liable to have heart affection, and that females to a greater extent than males are exposed to this complication.

We may now be permitted to record the particulars of the following thirteen cases.

CASE 1.—Emma L—, æt, 17, was admitted into the London Hospital December 20th, 1867. Her illness commenced eight days before admission with pain in all her limbs. This was her first attack of rheumatic fever. On admission she had a good deal of pain in several joints. The temperature in the axilla was 104°, pulse 144, respiration 44.

On the second day of admission the pain continued; she perspired profusely, and the rheumatic odour was well marked. The temperature was 103·2°, pulse 124, and respiration 40.

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[1] Vol. xii, third series, 1866; *vide* Cases 1, 2, 3, 5, 7, 8. Vol. xi, 1865; *vide* Dr. G. O. Rees' cases 1, 3, 4, also Dr. Gull's cases 1, 2, males, and 8, females.

On the third day, the 22nd December, the temperature was—

102·6° in the morning. Pulse 138. Respiration 44.
103·5° ,, evening. ,, 132. ,, 36.

23rd.—Fourth day of admission, had pain in the chest, otherwise free; could turn in bed without any difficulty; tongue almost clean; perspired freely; urine clear, acid.

Temperature	101·8°	morning.	Pulse 120.	Respiration 20.
,,	102°	evening.	,, 138.	,, —
24th, 5th day,	102·4°	morning.	,, 120.	,, 36.
25th, 6th day,	99·2°	morning.	,, 120.	,, 36.
,, ,,	100°	evening.	,, 100.	,, 32.
26th, 7th day,	98·4°		,, 100.	,, 36.
27th, 8th day,	98°		,, 100.	,, 32.
28th, 9th day,	97°		,, 90.	,, 24.
29th, 10th day,	97°		,, 80.	,, —

On the seventh day from admission, and the fifteenth of her illness, the temperature was normal; the pulse varied from 92 to 100, respiration from 22 to 36.

On the eleventh day, and nineteenth day of her illness, the temperature had remained normal for four days. Her pulse was 80, and the respiration had fallen to 24. She was in no pain and her tongue was clean, and she continued from this time to do well. The acute symptoms are calculated to have terminated on the eleventh day, but the temperature was normal on the seventh day, and continued so from that day, and this might be considered as the termination of the acute symptoms. The patient was not, however, entirely free from pain until the eleventh day, and the acute symptoms, therefore, are estimated to have ceased on the eleventh day. The total duration of her symptoms, dating from the time that her illness commenced, that is, eight days before admission, to the cessation of acute symptoms, was ten days.

Heart.—The heart's action was rapid and the impulse beat was unduly marked, but there was no abnormal sound throughout her illness.

Case 2.—Catherine C—, æt. 24, admitted into the London Hospital December 28th, 1867. This was her first attack of rheumatic fever. Her illness commenced five days before admission with pain in the left foot. She was very thirsty and had lost her appetite. On admission she complained of pain in her feet and knees. Her joints were somewhat swollen, hot, and red; her tongue was white; her appetite was bad, her face flushed, and her throat was sore; pharynx congested, but there was no ulceration. The temperature was 102·4°, pulse 120, respiration 36. Ordered diet of milk and beef tea, and an ounce of mint water three times a day.

On the 29th, the second day of admission, had slept pretty well. Pain in the right wrist in addition to the joints previously affected. Temperature 101°, pulse 116, respiration 27. Ordered ten grains of Dover's powder at bedtime.

30th.—Third day. Slept well. Pain in left shoulder, also a little pain in right knee and ankle; perspiring. Urine sp. gr. 1030, slightly acid.

Temperature	100·5° morning.	Pulse 100.	Respiration 40.
,,	101·6° evening.	,, 104.	,, 32.
31st, 4th day,	100·4° morning.	,, 100.	,, 24.
,, ,,	102·5° evening.	,, 112.	,, 36.

January 1st.—Fifth day, she was in much less pain.

Temperature	100·2° morning.	Pulse 104.	
,,	100·6° evening.	,, 108.	
2nd, 6th day,	100·2°	,, 92.	
3rd, 7th day,	99·2°	,, 84.	Respiration 24.

She was completely free from pain, excepting a little in the left shoulder-joint, but so little that she could move her arm freely; her tongue was clean and moist, and she did not perspire so much. Her pulse was normal, having fallen from 120 to 72; her respirations from 36 to 20. The temperature had fallen from 102° to 99·2° and 99·5°; it was therefore almost normal.

6th.—On the tenth day the temperature was 98·4°, pulse 72, respiration 20. On the eleventh day of admission the temperature was 98·4°, pulse 60, respiration 20. The temperature was carefully taken during the five following days, and it never exceeded 98·5°. The acute symptoms are calculated to have ended on the eleventh day. The total duration of her acute symptoms, including the five days before admission, was sixteen days. She was discharged February 7th.

Heart.—On the day of admission the cardiac dulness was normal; over both apex and base the first sound was prolonged. On the third day the first sound was still prolonged. On the fifth day it was prolonged. On the seventh day there was a well-marked systolic bruit over the third interspace close to the sternum, and the first sound was prolonged at the apex. On the ninth day the cardiac dulness was normal, and a feebly marked systolic bruit was heard over the base; the first sound at the apex was prolonged. On the seventeenth day the murmur was still faintly heard.

February 1st.—On the thirty-second day the cardiac dulness was normal, action quiet, and there was no abnormal sound.

Case 3.—Alfred W—, æt. 17, was admitted into the London Hospital October 25th, 1867. This was his first attack of rheumatic fever. His illness commenced six days before admission with pain and swelling in his left knee and ankle, and with thirst and loss of appetite. On admission he complained of great pain in his joints; his face was flushed, and he was very thirsty. The temperature in axilla was 102·4°, pulse 92, respiration 28.

On the second day of admission he could not turn in bed; there was great pain in the right ankle and in the wrists. He complained of sore throat, and some cheesy deposits were noticed on the tonsils. Temperature was 100°, pulse 104, respiration 36. An ounce of mint water every four hours was ordered, and milk and beef tea for diet.

On the 27th, the third day, he could not move in bed on account of the pain in his joints. Tongue was dry in the centre; urine acid, sp. gr. 1025;

passed fifty ounces in twenty-four hours. Temperature 102·8° morning, 102·4° evening; pulse 80, respiration 28.

On 28th, fourth day, pain, redness, and a little swelling in wrists, right knee, and ankle. Temperature 102·4° morning, 102° evening.

On 29th, fifth day, temperature 102° morning, 101° evening.

On 30th, sixth day, not in so much pain; sweating; rheumatic odour strongly marked. Temperature 101° morning, 104° evening. Ordered half a grain of opium every four hours during the night only.

On 31st, seventh day, he had slept better. sweating profusely. Temperature 100·8° morning, 101° evening. He was ordered half a grain of opium night and morning.

November 1st.—Felt better; in less pain; tongue clean; urine, passed thirty-five ounces in twenty-four hours, acid, sp. gr. 1020. Temperature 100° morning and evening.

2nd, ninth day, temperature 98·6° morning, 99·2° evening. Felt better, in much less pain.

3rd, tenth day, temperature 99° morning, 99·8° evening.

4th, eleventh day, temperature was normal, 98·4° morning, 98·5° evening.

5th, twelfth day, free from pain; tongue clean; urine acid, natural colour, passed thirty-five ounces in twenty-four hours, sp. gr. 1018.

8th.—On the fifteenth day he was out of bed, free from pain; appetite good, and placed on middle diet.

The acute symptoms had very greatly subsided on ninth day, and they continued to do so, and on the twelfth day they had completely subsided. The total duration of the acute symptoms, including the six days before admission, was eighteen days.

On the twentieth day he had continued to do well, and was free from pain. Discharged December 3rd, on the fortieth day.

Condition of the heart.—On the day of admission the apex impulse was unduly marked, there was no murmur, and the cardiac dulness was normal. On the 27th, the third day, the first sound was markedly prolonged under the left nipple. Over the third left costal cartilage it was so prolonged that it might fairly be called a slight systolic bruit. Cardiac dulness extended to the third rib; impulse was unduly marked. On the fifth day heart in same condition. On the eighth day the bruit was still heard at the base and also at the apex, but loudest at the base. On the twelfth day the rhythm was irregular and the bruit was still heard. On the fifteenth day the bruit was doubtful. December 3rd, when discharged, his heart was carefully examined; there was no abnormal sound; the cardiac dulness was normal; the apex-beat was in its normal position. The heart was therefore, as far as could be appreciated, healthy.

Case 4.—George M—, æt. 13, admitted into the London Hospital January 17th, 1868. This was his first attack of rheumatic fever. His illness commenced five days before admission with pain in his feet; his joints were swollen; he was thirsty and sweated a good deal. On the day of admission the rheumatic odour was well marked. He was thirsty, his appetite was bad, and there were pain and redness of his shoulder-joints and hands. His urine was acid, the specific gravity 1030; temperature 100·6°, pulse 80, respiration 28. On the second day of admission the temperature had fallen to 98·8°. On the third day he was free from pain, his tongue was moist and slightly coated. On the fourth day he was much better and in no pain, but the joints were a little stiff. The acute symptoms had entirely subsided on the fourth day, and it might be said on the third day.

The condition of the heart.—The total duration of the acute symptoms, including the five days before admission, was nine days. There was a systolic, apparently an aortic, bruit over the heart on admission, which remained when the patient was discharged.

Case 5.—Rebecca D—, æt. 18, admitted into the London Hospital January 28th, 1868. This was her first attack of rheumatic fever. Her illness commenced one day before admission with pain in the feet. On the day of admission her wrists were red and swollen, as well as the ankles. She perspired profusely. Her cheeks were flushed, and she was very thirsty. The urine was acid, and the specific gravity was 1030; the temperature 102·2°, her pulse 120, respiration 28.

On the 29th, second day of admission, the temperature was—
102·2° in the morning. Pulse 114. Respiration 28.
101·8° in the evening. „ 124. „ 32.
30th, the third day of admission, the temperature was—
102·6° in the morning. Pulse 124. Respiration 40.
102·6° in the evening. „ 128. „ 36.
31st, the fourth day of admission, the temperature was—
102° in the morning. Pulse 116. Respiration 32.
102·4° in the evening. „ 120. „ 32.
February 1st, the fifth day, the temperature was—
102·6° in the morning. Pulse 112. Respiration 32.
101·2° in the evening. „ 112. „ 28.
Pain and redness in the left wrist, but nowhere else. Skin perspiring and covered with miliaria.

2nd, the sixth day, pain in right wrist; urine faintly acid, sp. gr. 1014; temperature was—
101·4° in the morning. Pulse 96. Respiration 28.
101·2° in the evening. „ 92. „ 30.
3rd, the seventh day, sweated profusely, copious miliaria, pain in wrist only; urine was neutral, sp. gr. 1010, contained lithates; temperature was—
102° in the morning. Pulse 88. Respiration 24.
101·6 in the evening. „ 100. „ 28.
4th, the eighth day, the temperature was—
99·2° in the morning. Pulse 80. Respiration 20.
99° in the evening „ 72. „ 28.
5th, the ninth day, the temperature was—
99° in the morning. Pulse 84. Respiration 24.
100° in the evening. „ 88. „ 24.

6th, the tenth day, the temperature was—
99·2° in the morning. Pulse 80. Respiration 24.
99·4° in the evening. ,, 88. ,, 28.
7th, the eleventh day, the temperature was—
98·6° in the morning. Pulse 88. Respiration 26.
98·6° in the evening. ,, 88. ,, 24.

On the 4th she was in no pain, appetite was good, tongue was almost clean. Urine acid, sp. gr. 1015. Judging from these symptoms and the temperature, the acute symptoms appear to have ended on this day, *i. e.* the eighth day of admission. On the eleventh day she was quite convalescent; temperature normal. The total duration of the acute symptoms, including the day she was ill before admission, was nine days. She continued to do well and was discharged.

State of the heart.—On the day of admission there was diminished resonance as high as the second interspace; marked dulness as high as the third rib, and the dulness was bounded below by the fifth interspace; the apex-beat was unduly marked in the fourth interspace, and a little to the right of the nipple. In the third left interspace, close to the sternum, the first sound was prolonged, and at the apex also. On the fourth day of admission the first sound over the base of the heart resembled a faint bruit. On the fifth day it was a decided bruit, audible both at the base and apex. On the fourteenth day a systolic bruit was heard over the apex, accompanied by a thrill; and when the patient was discharged the systolic bruit over the apex remained.

Case 6.—Elizabeth W—, æt. 14, admitted into the London Hospital August 22nd, 1867. Her first attack of rheumatic fever. Suffered with an aching sensation in her leg fourteen days before admission; seven days before admission she had pains in the legs and hands. Two days afterwards she was laid up with pain and swelling in ankles and knees, and she remained in much the same state until she entered the hospital. On the day of admission she had pain, redness, and some swelling in her ankles and knees. Slight pain in the arms, tongue furred. Temperature was 102·4°, pulse 120, respiration 36. Ordered one ounce of mint water every four hours, five grains of Dover's powder every night at bedtime. Diet, milk and beef tea.

23rd, on the second day, the temperature was—
101·6° in the morning. Pulse 116. Respiration 36.
103·6° in the evening. ,, 116. ,, 44.
24th, third day, the temperature was—
100·6° in the morning. Pulse 104. Respiration 36.
101·4° in the evening. ,, 104. ,, 44.

The Dover's powder was discontinued.

25th, fourth day, pain and swelling in the left wrist; perspiring; temperature 100°, pulse 92, respiration 34.

26th, fifth day, not in so much pain; temperature 99°, pulse 84, respiration 26.

27th, sixth day, free from pain; temperature 98°, pulse 72, respiration 32.

28th, seventh day, in no pain; appetite good; tongue clean; skin cool, and not perspiring. The acute symptoms terminated on the sixth day. The total duration of acute symptoms, including the seven days before admission, was thirteen days. She was discharged on September 20th.

State of heart.—On the day of admission there was absolute dulness as high as the upper margin of the left third rib, and diminished resonance as high as the second left costal cartilage. The area of dulness was oval-shaped. It was bounded on the left by a vertical line through the nipple, and on the right by the left margin of the sternum, and below by the fifth rib. The first sound over the apex of the heart was short and sharp. Over the third left costal cartilage it was prolonged, and over the second left costal cartilage it was a decided but feeble systolic bruit; it was only heard over this spot. No thrill could be felt. On the third day of admission the bruit was heard all over the base of the heart. Cardiac dulness the same as before. On the fourth day the apex-beat was unduly marked in the fourth and fifth interspaces, and the systolic bruit was very distinctly heard over the second interspace, louder on the left than on the right. The bruit was not heard at the apex, and not conducted along the aorta. On the seventh day of admission there was a faint systolic bruit over the left second interspace, close to the sternum. The bruit was very localised, and not so distinctly heard as it was three days ago. On the twentieth day of admission there was still a slight systolic bruit over the base. Heart otherwise healthy. On the twenty-third the heart was normal, excepting that there was still heard a faint systolic bruit. It was more like a prolonged first sound than a bruit. When discharged, this abnormal character of first sound was faint and not well marked.

Case 7.—Mary Anne N—, æt. 21. Admitted into the London Hospital December 24th, 1867. Her first attack of rheumatic fever. Good health until twelve days before admission. At that time was seized with pain and swelling in her wrists and shoulders. Eight days before admission was obliged to go to bed, not able to stand on account of the pain in her legs, and was confined to bed until the day she was admitted into the hospital. On the day of admission she had pain in her knees, shoulders, and wrists. Her tongue was coated with yellow fur; her face was flushed, and she perspired freely. Temperature was 101·6°, pulse 124, respiration 28. Ordered diet of milk, beef tea, and lemonade, and an ounce of mint water three times a day.

25th, the second day of admission, the temperature was—
100·4° in the morning. Pulse 112. Respiration 24.
101° in the evening. ,, 112. ,, 28.
26th, the third day, the temperature was—
101·8° in the morning. Pulse 108. Respiration 20.
101·2° in the evening. ,, 108. ,, 28.

Complained of pain in the chest and sore throat. Sibilant respiration heard over front of the chest.

27th, the fourth day, pain in left knee, shoulder, and wrist. Sweated freely. Rheumatic odour was well marked. Temperature was—

99·4° in the morning. Pulse 108. Respiration 28.
101° in the evening. „ 108. „ 28.

Ordered linseed-meal poultice to be applied to the chest.

28th, the fifth day, ordered half an ounce of castor oil to be taken immediately, and a quarter of a grain of morphia at bedtime. Temperature was—

99° in the morning. Pulse 104. Respiration 24.
99·8° in the evening. „ 108. „ 28.

29th, sixth day, pain equally severe in left shoulder and right knee as it was three days ago. Tongue was clean, appetite much better; perspired freely; temperature was—

100° in the morning. Pulse 104. Respiration 24.
98·4° in the evening. „ 108. „ 24.

31st, eighth day, she was in no pain excepting in the left shoulder; temperature was—

100·2° in the morning. Pulse 104. Respiration 24.
100·6° in the evening. „ 108. „ 24.

January 1st, ninth day, temperature was—

99° in the morning. Pulse 88. Respiration 16.
101° in the evening. „ 96. „ 20.

2nd, tenth day, temperature was—

98·5° in the morning. Pulse 104. Respiration 24.
100·2° in the evening. „ 96. „ 24.

3rd, eleventh day, temperature was—

100·4° in the evening. Pulse 92. Respiration 20.

Free from pain.

4th, the twelfth day, temperature was—

98·8° in the morning. Pulse 88. Respiration 18.
99·5° in the evening. „ 96. „ 24.

5th, thirteenth day, temperature was—

98·4° in the morning. Pulse 80. Respiration 24.
99·5° in the evening. „ 80. „ 24.

6th, fourteenth day, temperature was—

98·4° in the morning. Pulse 80. Respiration 24.
99·5° in the evening. „ 84. „ 28.

7th, fifteenth day, temperature was—

98·8° in the morning. Pulse 76. Respiration 24.
100° in the evening. „ 92. „ 20.

8th, sixteenth day, temperature was—

98·6° in the morning. Pulse 72. Respiration 24.
99·2° in the evening. „ 88. „ 20.

9th, seventeenth day, temperature was—

99° in the morning. Pulse 72. Respiration 24.
99° in the evening. „ 68. „ 24.

10th, eighteenth day, temperature was—

99° in the morning. Pulse 68. Respiration 24.
99° in the evening. „ 80. „ 24.

11th, nineteenth day, temperature was—
98·4° in the morning. Pulse 80. Respiration 24.
98·2° in the evening. „ 78. „ 24.
12th, twentieth day, temperature was—
98° in the morning. Pulse 68. Respiration 24.
98·6° in the evening. „ 84. „ 20.
13th, twenty-first day, temperature was—
98° in the morning. Pulse 72. Respiration 24.
98·6° in the evening. „ 88. „ 24.

14th, twenty-second day, temperature was 98°, pulse 72, respiration 24, and she was out of bed, doing well.

In this case the temperature was normal or almost normal, not exceeding 99·5° on the twelfth day.

On fifteenth day it was normal in the morning and 100° in the evening. On sixteenth day it was normal, and remained so.

We may therefore, perhaps, fairly conclude that this patient was convalescent on fifteenth day, and it is to be noticed that she was totally free from pain on eleventh day, and continued free from it. The total duration of acute symptoms, including the twelve days before admission, was twenty-eight days.

Condition of the heart.—On admission cardiac dulness was normal, apex impulse unduly marked; there was a systolic bruit over the third left inter-space, close to the sternum. The bruit was not conducted along the aorta. On 22nd, fourth day, cardiac dulness extended as high as the second left costal cartilage, oval-shaped. Bruit the same as before. 29th, sixth day, heart in the same condition. Thirteenth day, cardiac dulness was normal, and no bruit was heard.

On twenty-third day the heart was normal.

Case 8.—Jessie C—, æt. 18, admitted October 29th, 1867. Her first attack of rheumatic fever. Eleven days before admission she had pains in her limbs; four days before, she was obliged to go to bed. On the day of admission complained of pain in her thighs, knees, ankles, and left arm. Appetite bad; tongue red in the centre and furred at the sides; temperature 100·4°, pulse 100, respiration 24. Ordered 1 oz. mint water every four hours, ¼ grain morphia at bedtime. Milk and beef tea for diet.

30th, second day, temperature 101°, pulse 92, respiration 24; was in great pain; perspired very much.

31st, third day, temperature 101·4°, pulse 100, respiration 32.

November 1st, fourth day, was in more pain, which was great in left leg; redness and swelling in left wrist; perspired a great deal; urine acid, contained no albumen, sp. gr. 1021. Temperature—
102° morning. Pulse 76. Respiration 24.
101·8° evening. „ 72. „ 32.
Ordered ½ grain opium twice a day to relieve the great pain.

2nd, fifth day, unable to move in bed; in great pain. Temperature—
101·4° morning. Pulse 98. Respiration 22.
102° evening. „ 108. „ 30.

3rd.—Temperature 102·2°, pulse 108, respiration 28. Ordered ½ oz. castor oil.

4th, seventh day. Temperature—

101·8° morning. Pulse 102. Respiration 24.
102·8° evening. ,, 100. ,, 20.

5th, eighth day, suffered great pain. Temperature—

102·4° morning. Pulse 104. Respiration 20.
102·3° evening. ,, 120. ,, 32.

Ordered 1 grain opium every four hours.

6th, ninth day, temperature 102°, pulse 116, respiration 30.

7th, tenth day, great pain in shoulder; less pain in legs; perspired freely. Temperature—

102·2° morning. Pulse 108. Respiration 36.
102·4° evening. ,, 112. ,, 32.

8th, eleventh day.—She complained very much of pain in her joints. Temperature—

103° morning. Pulse 120. Respiration 30.
102·4° evening. ,, 112. ,, 28.

Ordered 1 grain opium twice a day. Temperature remained at 100° until November 14th, the seventeenth day, when the pain was much less. Temperature—

99° morning. Pulse 72. Respiration 20.
99° evening. ,, 80. ,, 22.

15th, eighteenth day. Temperature—

98° morning. Pulse 80. Respiration 18.
98·4° evening. ,, 80. ,, 18.

17th, twentieth day, was free from pain; temperature normal; appetite returning.

27th, thirtieth day, had had no return of the pain; appetite was good; tongue clean; skin cool; was anxious to get up.

December 5th.—She was quite well and discharged. The acute symptoms had undoubtedly ceased on the nineteenth or twentieth day.

The total duration of acute symptoms, including eleven days before admission, was thirty-one days.

Condition of heart.—On the day of admission a systolic bruit was heard. On the third day there was a harsh bruit at the base; cardiac dulness reached as high as the second interspace; systolic bruit was heard over the base. On the tenth day this bruit was heard also at the apex. On the thirty-first day, November 28th, the dulness was normal, first sound feeble; second sound unduly marked, but there was no abnormal bruit.

Case 9.—Rachel L—, æt. 12, admitted into the London Hospital October 29th, 1867. Six weeks before admission she was laid up a fortnight with pain in her limbs; subsequently she improved; the pain afterwards returned, and she was very weak. On the day of admission she was very anæmic-looking, and had little pain in her joints. On the second day of admission temperature 101°, pulse 102, respiration 38. On the third day the temperature was 101°, pulse 102; the tongue was slightly coated, and she had pain in both knees. On the fifth day she was free from pain, the temperature was normal, and she continued to do well. The acute symptoms in this case had terminated by the fifth day. The arthritis was not severe. The total duration of the acute symptoms, including the days she was ill before admission into the hospital, could not be calculated.

State of the heart.—The cardiac dulness was increased, and reached as high as the second costal cartilage. Over the base a thrill was felt, and a superficial creaking sound accompanied both sounds of the heart, apparently the result of pericarditis. There was a systolic bruit at the apex on admission, and when she was convalescent there was a well-marked systolic bruit over the apex of the heart which was conducted along the axilla.

CASE 10.—Annie A—, æt. 15, admitted October 25th, 1867. Her first attack of rheumatic fever. She had suffered from pain in her knees and back ten days before admission. She was confined to bed only two days before admission. The day she entered the hospital the temperature was 102·4°, pulse was 128, respiration 28. She had pain in several joints. Her tongue was furred; the urine was acid, and its specific gravity was 1035. Ordered a mixture containing a little coloured syrup and water every four hours.

26th, second day, temperature—
101·6° morning. Pulse 128. Respiration 32.
102·4° evening. ,, 120. ,, 34.
27th, third day, temperature—
101° morning. Pulse 116. Respiration 28.
101·4° evening. ,, 120. ,, 28.
28th, fourth day, temperature—
101° morning. Pulse 116. Respiration 20.
102·6° evening. ,, 129. ,, 28.
Ordered 1 grain opium to be taken at bedtime.
29th, fifth day, temperature—
103° morning. Pulse 120. Respiration 30.
101° evening. ,, 120. ,, 36.
30th, sixth day, pain in both hands. Temperature—
101·4° morning. Pulse 120. Respiration 28.
100·6° evening. ,, 116. ,, 32.
31st, seventh day, temperature—
101·4° morning. Pulse 112. Respiration 30.
102° evening. ,, 116. ,, 24.

November 1st, eighth day, in no pain; her limbs felt stiff. Ordered ½ oz. castor oil. Temperature—
101·2° morning. Pulse 96. Respiration 22.
100° evening. ,, 112. ,, 20.

2nd, ninth day.—Felt better; in no pain, except a little in right shoulder; urine scanty, acid, sp. gr. 1032. Temperature—
101° morning. Pulse 112. Respiration 20.
100·4° evening. ,, 112. ,, 24.

3rd, tenth day, temperature—
98·6° morning. Pulse 112. Respiration 32.
100·2° evening. „ 110. „ 28.
4th, eleventh day, temperature—
99·6° morning. Pulse 120. Respiration 30.
99·8° evening. „ 96. „ 24.

5th, twelfth day.—She had been free from pain three days; her appetite was good; she did not perspire, and her tongue was clean. Sp. gr. of urine 1026. Temperature—
98° morning. Pulse 88. Respiration 26.
99° evening. „ 96. „ 20.

7th, fourteenth day.—In no pain; appetite good. Temperature—
98° morning. Pulse 80. Respiration 20.
99·6° evening. „ 88. „ 28.

She continued to do well, and had no return of pain.

13th, twentieth day.—Out of bed.

On 27th, thirty-fourth day of admission, discharged.

In this case the acute symptoms had completely disappeared on the twelfth day. The total duration of acute symptoms, including the time that she was ill before coming into the hospital, might be calculated at twenty-two days, or perhaps, more correctly speaking, at thirteen days.

Condition of the heart.—On admission a systolic bruit was heard over the base of the heart; apex-beat diffused. On the 30th October it was still heard. November 1st, a systolic bruit was heard under the left nipple. When discharged the murmur was still heard at the apex and at the angle of the left scapula.

CASE 11.—Annie D—, æt. 14, was admitted into the London Hospital October 25th, 1868. Her first attack of rheumatic fever. Her illness commenced three days before admission with pain in her right leg. The day before admission she was compelled to lie up. The day she came into the hospital there was pain in both knees and shoulders; the knees were red and swollen. On the second day the temperature was 101·2°, pulse 124, respiration 32. On the third day the temperature was 99° and 99·8°, pulse 104, respiration 24. On the fourth day she was in less pain, and temperature was normal. On the seventh day she was free from pain; her tongue was clean; the temperature was normal; the urine was slightly acid, its specific gravity 1019. After this she rapidly improved.

Here the acute symptoms had ceased on the seventh day, and the total duration of acute symptoms, including three days before admission, was ten days.

Condition of the heart.—There was a systolic bruit at the base on admission and when discharged from the hospital.

CASE 12.—Henry K—, æt. 17, admitted into the London Hospital January 11th, 1868. His first attack of rheumatic fever. His illness commenced three weeks before admission with pain in his limbs. On the day of admission he had pain and swelling in the right wrist and shoulder; perspiring freely; temperature 100·5°, pulse 112, respiration 20. Three days after this he was free from pain. Temperature 98·8°, pulse 84, respiration 16, and he continued to do well.

Condition of the heart.—On the 13th January, the third day of admission (the state of the heart is not mentioned in the report-book until the 13th), the heart's impulse was unduly marked and a bruit was heard over the apex.

16th.—The murmur was heard over the base and apex, but very faintly heard over the apex.

20th.—No abnormal sound was heard over the heart.

The acute symptoms had ended in this case on the third day of admission, and the total duration of the rheumatic symptoms, calculating the twenty-one days before admission, was twenty-four days.

Case 13.—Sarah B—, æt. 16, admitted into the London Hospital January 10th, 1867. Her first attack of rheumatic fever. Four days before admission had pains in her thighs, which extended to her knees and feet.

On the 11th and 12th, the second and third days of admission, there were no decided rheumatic symptoms. There was no visible affection in any joint; she could stand and move about without any apparent pain or discomfort.

13th, the fourth day of admission.—There was much pain and slight swelling in the ankles.

14th, fifth day.—The affection of the joints was still more marked.

17th, eighth day.—Pain in both wrists, and on the left side.

18th, ninth day.—Pains in both elbows, no pain in any other joint; skin hot and dry; respiration 48, pulse 120.

19th, tenth day.—Pain in both wrists; urine, copious lithates, sp. gr. 1035, acid and scanty; perspired freely.

20th, eleventh day.—Great pain in the region of the heart. Not much pain in her joints. Temperature 102·8°, respiration 48, pulse 120.

22nd, thirteenth day.—Almost free from pain. Temperature 102·5°, respiration 52, pulse 126. Urine 18 oz. in twenty-four hours, sp. gr. 1029, loaded with lithates. She continued much the same, the temperature a little over 102°, the pulse about 114, and respiration about 40. One day free from pain, the next day pain in all her joints.

30th, twenty-first day.—In no pain; urine 20 oz. in twenty-four hours sp. gr. 1020, with lithates, and her temperature had fallen to 98°. She continued to improve.

February 2nd, twenty-fourth day.—Her temperature was normal, and had remained so since the 30th January. Urine 23 oz. in twenty-four hours, sp. gr. 1015. From this time she steadily improved, and was discharged cured.

On the twenty-first day of admission the acute symptoms are calculated to have ended. The total duration of the acute symptoms, including the four days before admission, was twenty-five days.

Condition of the heart.—On the day of admission there was no abnormal bruit, but the first sound was prolonged and very feeble. The cardiac dulness was normal. On the sixth day after admission, and two days after the rheumatic affection of the joints became well marked, a faint systolic bruit was heard all over the cardiac region, and the cardiac dulness was slightly increased. On the eighth day of admission the cardiac dulness was increased and a harsh to-and-fro murmur was heard over and about the apex of the heart. On the ninth day the cardiac dulness reached as high as the second interspace. The impulse was diffused all over the cardiac region, and a to-and-fro murmur was heard. On the eleventh day a faint to-and-fro murmur was heard all over the cardiac region. There was dulness. The tactile vocal fremitus was distinct. There were bronchophony, bronchial breathing, and fine crepitation during deep inspirations over the lower portion of the left chest posteriorly. At this period there was well-marked pneumonia and pericarditis. On the thirteenth day the to-and-fro murmur was less distinct. The dulness, bronchial breathing, and bronchophony were very distinct over the lower part of left lung. On the fourteenth day the heart's sounds were normal. On the sixteenth day the heart's sounds were normal, and the bronchial breathing and the bronchophony were not so distinct. On the seventeenth day the heart's sounds were normal, and the bronchial breathing and bronchophony were still heard. On the twentieth day of admission no abnormal sounds of the heart; coarse crepitation over the lower part of the left lung. On the twenty-first day the physical signs over the left lung were much less distinct, and they gradually disappeared. When this patient was discharged no abnormal sounds were heard over the region of the heart or lungs.

The acute symptoms terminated in these cases respectively on the 11th, 12th, 4th, 8th, 6th, 15th, 20th, 5th, 12th, 7th, 21st, and 3rd days. The average duration of the acute symptoms, therefore, in the thirteen cases was ten days.

In the first series of cases under the care of Dr. Gull, treated for the most part with mint water only, which we have published in vol. xi, 3rd series, of 'Guy's Hospital Reports,' 1865, the average duration of the acute symptoms was 8·5 days.

In the second series, published in vol. xii, 3rd series of the 'Guy's Hospital Reports' of the year 1866,[1] the average duration of the acute symptoms was 9 days.

And in this our third series of cases, as we have stated above, the average duration was 10 days.

There is, therefore, a difference of one day and a half only between the three series of cases, the average of these three being 9·1 days.

This, we believe, will usually be found to be about the length of time that the acute symptoms generally extend over in cases of rheumatic fever, where there is no severe heart disease.

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[1] As this paper bears the name of Dr. Sutton only it has not been reprinted in this volume.

In the third series of cases, as we have just stated, the average duration of the acute symptoms was calculated at 10 days, whereas in our first series of cases it was 8·5 days. This difference may be explained when we state that much greater care was taken in estimating the duration of the acute symptoms in the third series of cases than in the first. In the third series the temperature was taken every morning and evening, and until this was found to be normal the acute symptoms were not considered to have ceased. It not infrequently happened that the tongue was clean and the patient was free from pain, but the temperature was higher than normal; in such cases, guided by the thermometer, we have concluded that the acute symptoms had not terminated; whereas in the first and second series of cases, where the thermometer was not used, when the tongue was clean, the skin felt cool, and the patient was free from pain, the acute symptoms were regarded as having ceased. Had the temperature been taken in the first and second class of cases, we believe the average duration of the acute symptoms would have been about 10 days instead of 8·5 days.

The total duration of the acute symptoms, including the time that the patient was ill before coming into the hospital, was respectively 19, 15, 18, 9, 9, 14, 28, 31, 22, 10, 24, and 25 days, and the total duration of the acute symptoms—that is, from their commencement to their cessation—was, on an average, 19 days.

We now proceed to ask—Is the duration of rheumatic fever longer when it is treated on the expectant plan, or when it is treated by drugs? We very much regret that, owing to the arrangement of Dr. Dickinson's paper (*vide* Royal Medical and Chirurgical Society's 'Transactions,' vol. xlv, on "The Treatment of Rheumatic Fever"), we could not compare his cases with our own. In Dr. Garrod's cases (Royal Med. and Chir. Soc. 'Transactions,' vol. xxxviii), treated by large doses of alkali, the male patients were, on an average, in the hospital 6·2 days before the acute symptoms subsided, and females were 7·3 days before the acute symptoms subsided. The average of the two was 6·75 days.

In Dr. Herbert Davies' cases (*vide* 'London Hospital Reports,' vol. i), treated by blisters, the duration of the acute symptoms, while the patients were in the hospital, was, on an average, 8·4 days.

When, however, we take the thermometer as a guide, we find in Dr. Herbert Davies' cases that, although the patients were free from pain, and the joint symptoms had also subsided, on an average, in 8·4 days, yet the temperature of the body was not normal until, on an average, 9·5 days.

In Dr. G. Owen Rees' cases ('Guy's Hospital Reports,' vol. xii, 3rd series, 1866), treated by lemon-juice, the duration of the acute symptoms in the hospital was 6·8 days.

And under the expectant treatment the duration of the acute symptoms in the hospital was 9·1 days.

To give this evidence more briefly, under the full alkaline

treatment the average duration was .	6·75 days.
Lemon-juice ditto . .	6·8 "
Blister treatment ditto .	8·4 "
Expectant treatment ditto .	9·1 "

There was, therefore, a difference of a little more than two days in favour of the full alkaline and lemon-juice treatment, and the average in the blister and expectant treatment was almost equal.

When, however, the duration of the acute symptoms was tested still more accurately by the aid of the thermometer, we find that under the blister treatment it was, on an average, 9·5, under the expectant treatment 10 days.

The total duration of the acute symptoms, including the time the patients were ill before and after going into the hospital, was—

Under the full alkaline treatment .	13·5 days.
Under the blister treatment (not estimating by aid of the thermometer) was . . .	15·7 "
When the acute symptoms were estimated by the thermometer the duration was (under the blister treatment) . . .	17·27 "
Under the expectant treatment .	19·0 "

These differences are very slight, so much so that they appear to us to argue that one plan of treatment (as regards shortening the duration of the rheumatic process) has no great advantage over the other. Probably it will be admitted that no great importance should be attached to the fact that under one plan of treatment the duration of the acute symptoms was a day or two less, for every physician is well aware how difficult it is to fix the period when the acute symptoms have just ceased, so as not to be a day before or after the time when they have really subsided; for patients differ very much in estimating their own pain, and we therefore have to deal with all the uncertainty of subjective symptoms. It can, perhaps, be readily understood that one practitioner might consider a patient had lost all his acute symptoms to-day, whilst another practitioner, guided by the fact that the tongue was almost though not quite clean, that the patient was practically although not absolutely free from pain in the joints, might consider that the acute symptoms had not quite subsided, and then allow another day or two to pass over before he decided that the acute symptoms had ceased. Bearing this in mind, it appears to us that we cannot attach much importance to this difference of one or two days.

We therefore submit that there is no evidence to show conclusively that alkalies, lemon-juice, or blisters curtail the duration of the rheumatic process. And we agree with Dr. Barclay when (*vide* his work on 'Gout and Rheumatism,' p. 32), speaking of the administration of alkali in rheumatic fever, he states, "We are not yet in a position to say that it exercises any influence in curtailing the duration of the disease."

With respect to lemon-juice also, we are not satisfied that it has any such power.

We have witnessed the trial of Dr. Herbert Davies' blister treatment in a few cases which have occurred in Guy's Hospital, under the care of Dr. Wilks and the late Dr. Barlow, also in cases which have occurred in the London Hospital, and it has appeared to us to relieve very much the pain and sufferings of the patient in some cases, but it has not appeared to curtail the rheumatic process. We noticed that although the temperature of the body fell during the application of the blisters, yet it did not immediately return to its normal standard. It remained higher than normal for some days after the pain had abated, nor in several cases did the tongue become clean, the appetite return, nor the urine fall in specific gravity, as usually happens when the rheumatic process is really over. Moreover Dr. Davies' cases do not show, when compared with those here recorded, any decided evidence in favour of the blister treatment beyond the fact that the blisters appear to have given much and speedy relief to the pain in the joints. We therefore regard the application of blisters as a valuable agent in relieving the sufferings of the patient, but we are unable to say that it curtails the duration of the rheumatic process.

Our cases appear to us to teach that the rheumatic process runs its course under the expectant plan as favorably as under the treatment by drugs.

While it has been admitted by those who advocate the treatment of rheumatic fever by alkalies, that large doses of such drugs do not curtail the duration of the disease, nor bring very great relief to the patient, yet it is stated, to quote the words of Dr. Barclay, "that it does most incontestably prevent inflammation of the heart;" and he further states "that the proportion of heart ailments is very much reduced by the employment of the alkaline treatment."

We propose to inquire what evidence there is to support this statement.

We may now pass on to show what was the state of the heart in those cases where there was well-marked disease of that organ. In thirteen of these twenty-five cases there were physical signs indicating organic disease of the heart, particulars of which may be briefly given as follows:—It may be noticed that in every one of these cases the heart was more or less organically diseased when the patient was admitted into the hospital. In five of these thirteen cases there were physical signs of pericarditis on admission, and in three of the five there was a mitral bruit when the patients were discharged from the hospital. In a fourth a systolic aortic bruit was heard, and in the fifth case the heart was healthy when the patient was discharged. In one patient the first sound was prolonged at the base on admission, subsequently pericarditis and pneumonia supervened, and the heart was healthy when the patient was discharged from the hospital. In three patients there was a mitral murmur on admission and when discharged from the hospital. In one there was an aortic obstructive bruit on admission and when discharged. In one the cardiac dulness was normal; the first sound was prolonged at the base on admission, and a mitral bruit existed when the patient was discharged. In one case there was a systolic bruit at the base on admission, and at the apex when discharged. In one case the apex-beat was unduly marked, and there was præcordial pain on the day after admission; a new-leather-creaking bruit subsequently developed itself, and the patient died.

Besides these cases in which there was well-marked organic disease of the heart, there were twelve others in which there was no evidence of organic disease of the heart, but there was in nearly all more or less evidence of functional disturbance of this organ, or of changes in the blood producing so-called anæmic bruits. And in one or two cases the physical signs were such as might be said to indicate slight disease in the pericardium. These indications were, however, so slightly marked that it would be a matter of dispute as to whether they did or did not indicate morbid changes in the tissues of the heart. Not one of these patients had disease of the heart when discharged from the hospital.

The particulars of the twelve cases may be briefly summed up as follows:—In one case there was no bruit, but the heart's action was unduly marked. In one case there was a systolic, apparently an anæmic, bruit at the base on admission and when discharged. In one case the first sound was prolonged on the day of admission; on the seventh day a bruit was heard over the base, which afterwards entirely disappeared. In one the apex-beat was unduly marked on admission, and on the third day a bruit was heard, and the heart was healthy when discharged. In three cases the cardiac dulness was somewhat increased, and a systolic bruit was heard over the base when admitted, but it was indistinct when the patient was discharged in one of the three cases, and there was a systolic bruit at the base on admission, and the heart was healthy when discharged, in two of the three cases. In one case there was a systolic bruit at the base on admission, and the cardiac dulness was somewhat increased on the third day, and the heart was healthy when the patient was discharged. In one case a bruit was heard at the apex of the heart, which afterwards disappeared. In one a new-leather-creaking sound was heard, and the heart was healthy when discharged. In one the first sound was prolonged and the dulness increased on admission; afterwards a bruit was heard, and the heart was healthy when the patient was discharged. In one there was a bruit at the base on admission, and the first sound was prolonged when the patient was discharged from the hospital.

These details concerning the heart show that when rheumatic inflammation attacks the heart, and is allowed to run its natural course, the pericardium may become very extensively diseased, and yet the valves of the heart may escape, but in other cases in which there is pericarditis the valves may become diseased and rendered incompetent. Nor does it follow, because the pericarditis is severe and extensive, that the valves of the heart will certainly become diseased, but in the majority of cases in which there was severe pericarditis the valves did become involved.

Again, the cardiac dulness may be somewhat increased and extend higher than normal, and the first sound may be prolonged at the base. This prolonged first sound may develop into a bruit and be followed by well-marked mitral disease. In other cases there may be a similar increase of cardiac dulness, and a new-leather-creaking sound may be heard at the base of the heart, and this may subside and the heart remain healthy. Further, a prolonged first sound may be heard over the base of the heart, and this may develop into a systolic bruit and afterwards entirely disappear, leaving the heart healthy. A slight bruit may be heard over the apex of the heart, and afterwards it may completely disappear and the heart remain healthy.

It is instructive to notice that there was more or less modification of the heart sounds during some period of the attack in every case except in one. In only one case did the heart sounds remain perfectly healthy throughout the entire disease. But in eleven cases the abnormal sounds were such as are not usually considered to indicate any organic affection of that organ, and these abnormal sounds subsequently totally disappeared, and there was no evidence of cardiac disease when the patients were discharged from the hospital.

We now proceed to ask the question—If the heart was healthy when the patients were admitted into the hospital, was it common for it to become diseased while the patients were under treatment by mint water in the hospital?

Our cases show that when patients free from heart affection were admitted into the hospital suffering from rheumatic fever, and the disease was allowed to run its natural course uninfluenced by drugs, it was a very rare thing for the heart to become diseased while the patients were under treatment in the hospital.

In only one case where the sounds of the heart were healthy on admission did the heart become organically diseased while the patient was under treatment. This was a peculiar case (*vide* Case 8, 'Guy's Hospital Reports,' vol. xii, 3rd series, p. 528), such a one as is very rarely met with. The peculiarity of this case was that the patient died suddenly, and there was nothing found on post-mortem examination to account for her death, and the only evidence of heart disease was a very small quantity of recent lymph at the base of the heart.

The patient was a female æt. 25, who was admitted into Guy's Hospital under the care of Dr. Wilks. When first admitted she was suffering a good deal; she was in great distress, very restless and anxious. She complained very much of distress about her chest, and when questioned as to whether she had any pain there she answered, "It feels so tight, I can hardly breathe." On the day of admission Dr. Sutton listened to her heart and heard no abnormal sound; two days afterwards a new-leather-creaking sound was heard over the base of the heart.

This case was such an exceptional one that it might be fairly placed on one side, but even in this case there was some evidence of cardiac mischief within forty-eight hours after she was admitted into the hospital, as shown by the great distress the patient was suffering about the chest. And clinical experience has shown that severe distress and pain in the chest may be the first indication of pericarditis, and may precede the physical signs and exist without there being any physical signs to reveal the inflammation of the serous membrane.

In another case the indications of heart affection were very slight indeed when the patient was admitted into the hospital. A girl, æt. 16, was under the care of Dr. H. G. Sutton in the London Hospital (*vide* Case 13, Sarah B—, given in this paper, p. 526). On the day of admission she stated that she had pain in her ankles, but she could stand and walk apparently without any difficulty, and there was no redness, tenderness, or swelling of the joints. The cardiac dulness was normal, but the first sound of the heart over the base was prolonged and feeble. There was no bruit. She had, she said, felt pain in her legs for four days before coming into the hospital. After she had been in the hospital two days the rheumatic symptoms developed themselves, and after five days the cardiac dulness was slightly raised and a systolic bruit was faintly heard all over the cardiac region. After six days the heart's action was rapid, the apex-beat was unduly marked, and the systolic bruit was still heard. After seven days a to-and-fro murmur was heard over the base of the heart. From this time the pericarditis was well marked, and severe pneumonia also supervened. The patient had no evidence of heart disease when she left the hospital.

Unlike what usually happens with hospital patients suffering from rheumatic fever, the last-named patient was in the hospital from the commencement to the termination of her attack of rheumatic fever. When she was admitted the rheumatic process was so slightly marked that it was doubtful as to whether there was anything the matter with her. During the first two days that she was in the hospital there were no objective signs of rheumatic fever. After this time the rheumatic process gradually developed, and the heart disease was developed simultaneously with tho joint affection.

Ordinarily patients suffering from rheumatic fever do not enter the hospital until the disease has been going on some days, usually about six or eight, and in a great number of cases the heart has already become diseased before entering the hospital. If, however, the patients were in the hospital at the commencement of the disease, we should probably more frequently observe the heart become diseased while they were under treatment. One patient, therefore, that was in the hospital from the first to the last of the rheumatic fever ought not to be compared with the cases that have suffered from rheumatic fever for some days before coming under observation.

Our cases of rheumatic fever, therefore, show that of twenty-five patients suffering from a first attack of this disease, twelve had organic affection of the heart on admission into the hospital.

In one patient who was in the hospital from the commencement of the attack there was some, but very doubtful, evidence of heart disease on admission into the hospital, and pericarditis supervened while under treatment. In another patient there were no physical signs of heart disease on admission, but there was great distress and pain in the chest. Forty-eight hours after admission there were indications of pericarditis in this case. In eleven cases there was no organic disease of the heart on admission, and none supervened while the patients were under treatment.

Our cases further show that where there was no evidence of organic disease of the heart on admission into the hospital the heart was free from all evidence of disease when the patients were discharged from the hospital. Not one of our patients came into the hospital without heart disease and left the hospital with evidence showing that the heart was diseased.

The experience gained in these cases of rheumatic fever which were allowed to run their natural course uninfluenced by drugs tends to prove that, if patients are admitted into the hospital suffering from a first attack of rheumatic fever, and the heart is not diseased on admission, it will very rarely become organically diseased while the patient is under treatment.

It is rare for the heart to become diseased when the disease is allowed to run its course uninfluenced by drugs, and, judging from the evidence before the profession, it is also rare for the heart to become diseased when rheumatic fever is treated by drugs.

In support of the latter statement we may be permitted to bring forward the following evidence:—Dr. Garrod, speaking of the alkali treatment in rheumatic fever (*vide* Royal Medical and Chirurgical Society's 'Transactions,' vol. xxxviii, p. 151), says, "I cannot help thinking that an effect is likewise produced on the cardiac disease to a very considerable and important extent." In no case did the affection of the heart ensue after the patient had been more than forty-eight hours under the influence of medicine.

The statistics of Dr. Dickinson (*vide* Med. and Chir. Soc. 'Trans.,' vol. xlv, p. 350) show that in forty-eight patients subject to the full alkaline treatment only one had pericarditis commencing after treatment had begun.

Dr. Herbert Davies, speaking of the treatment of rheumatic fevers by blisters (*vide* 'London Hospital Reports,' vol. i, p. 300), says, "In no case where the heart was sound at the time of admission did any organic lession subsequently develop itself."

Dr. G. O. Rees also has informed us that when cases of rheumatic fever were treated with lemon-juice it was exceedingly rare to find the heart become diseased during treatment, provided it was healthy when the patient was admitted into the hospital.

Dr. Basham, in his paper on "The Treatment of Rheumatic Fever by Nitrate of Potash" (Med. and Chir. Soc. 'Trans.,' vol. xxxi, p. 14), gives, in a tabular form, an account of seventy-nine cases of this disease. One of these cases was probably not rheumatic fever, making the number seventy-eight cases, all of which were treated by nitrate of potash.

In sixty-one cases there was no heart disease. In twelve cases there was no heart disease on admission, and in only five cases did the heart become diseased while under treatment.

The experience, therefore, of these several physicians in cases of rheumatic fever is, that it is rare for the heart to become affected while patients are under treatment in the hospital; that in nearly all the cases where there was heart disease it had commenced before the patient was admitted into the hospital.

It is difficult to understand how remedies so different in their nature and in their actions should all work in such a manner as to bring about the same result—how the administration of large doses of alkali should produce the same result as the application of blisters to the joints.

It may be said that the operation of alkalies, lemon-juice, and blisters are different, but that they all accomplish the same end by either neutralising or eliminating the poison or the materies morbi of rheumatic fever. But can we reason in this way without begging the question? For it is not proved that there is a poison or materies morbi to be either eliminated or neutralised, and if we propose a theory to explain how these different remedies destroy the theoretical materies morbi it appears to us that we are only making a theory to explain a theory.

Seeing, therefore, that the heart rarely became affected when the rheumatic fever was not treated by drugs, we are compelled to ask the question—What evidence is there to show that any of the different plans of treatment which are advocated have any decided power in preventing the heart becoming diseased?

It appears to us that there is not sufficient evidence to prove that any of the systems of treatment have power to prevent the heart becoming diseased; and in concluding that the treatment has prevented the heart becoming diseased, physicians have overlooked the fact that there might be no tendency at the time that the patients were under treatment for the heart to become diseased, and our cases show that the good results which have been attributed to the influence of the remedies also occurred when no special remedies were used.

Our cases, therefore, tend to teach that these good results were due, not to the drugs, but to the natural course of the disease.

Before we conclude that it was something connected with the natural course of the disease which prevented the heart becoming diseased while the patients were under treatment in the hospital, it is necessary to ask, was there not something common to all these different plans of cure which prevented the heart becoming diseased, and was this something not present in the blister, in the alkali, the acid, and in the expectant plan of treatment?

The only things, perhaps, which were common to all these modes of treatment were rest and regulated diet.

Then the question arises—Are rest and regulated diet sufficient to explain this immunity from heart disease? It appears to us that they are not sufficient. For patients suffering from a primary attack of rheumatic fever have had well-marked symptoms of that disease for some days before coming into the hospital, and the heart has been organically diseased on admission, yet these patients have rested and lain in bed some days and even weeks before entering the hospital. Again, other patients have not rested while they were suffering pain in their joints and had other symptoms of rheumatic fever, and yet on admission into the hospital with a primary attack of rheumatic fever, the heart has been found to be free from disease.

It would appear, therefore, that rest and regulated diet are not alone sufficient to explain why the heart rarely becomes affected when the patients are under treatment in the hospital.

We shall now pass on to point out evidence which tends to show that when the heart becomes diseased in rheumatic fever it does so at an early stage of the disease, and if the heart does not become diseased during the first week of the rheumatic fever it rarely does so afterwards.

In order to do this it is necessary to show, when patients labouring under a primary attack of rheumatic fever were admitted into the hospital with heart disease, that such disease had set in during the first week of the fever.

We now beg to bring forward brief particulars of cases which show that the heart becomes involved during the early part of the attack.

In the case of Sarah B—, æt. 16 (Case 13, before referred to, pp. 526, 535), admitted into the London Hospital January 10th, 1867, four days previous to admission she had some pain in her ankles. During the first and second days of admission there were no decided rheumatic symptoms; she said her ankles were painful, but she could move freely in bed and could stand and walk about. Her tongue was clean, her skin cool, and it was very doubtful whether there were any indications of approaching rheumatic fever. Respecting the heart, there was no bruit, but the first sound was prolonged and feeble. The cardiac dulness was normal. On the third and fourth days of admission there were more decided rheumatic symptoms, pain in the left wrist and right ankle. On the fifth day the prolonged first sound mentioned above was found to have developed into a systolic bruit; this was heard all over the cardiac region. The cardiac dulness was slightly increased. On the eighth day of admission, and five days after the rheumatic symptoms were well marked, the systolic bruit was replaced by a harsh to-and-fro murmur, and the cardiac dulness was increased.

The second case was that of a female æt. 18 (*vide* Case 5 recorded in this paper, p. 518), who was admitted into the London Hospital January 28th, 1868. This was her first attack of rheumatic fever. Two days before admission she did not feel quite well, but there were no decided rheumatic symptoms until the day before she entered the hospital. On the second day of the rheumatic attack there was evidence showing that the heart was involved. The cardiac dulness reached as high as the third rib, and there was diminished resonance as high as the second rib.

The visible impulse was unduly marked over the third interspace close to the sternum; the first sound was prolonged and almost equal to a bruit, also prolonged over the apex. On the fifth day of admission the prolonged first sound resembled a faint bruit. On the sixth day it had become developed into a marked bruit, which was audible both over the base and apex of the heart; this murmur subsequently became developed into a well-marked mitral bruit, which was present when she left the hospital.

In a third case, a female æt. 14 (*vide* Case 11 in this paper, p. 525) was admitted into the London Hospital October 25th, 1868. This was her first attack of rheumatic fever, and she stated that she had never been ill before. The rheumatic symptoms appeared three days before admission. October

26th, the second day of admission and fifth of the rheumatic process, there was evidence showing that the heart was involved. The heart's impulse was diffused and visible in the second and third interspace; the cardiac dulness reached as high as the second costal cartilage and as low as the sixth rib; a well-marked, apparently an aortic, systolic bruit was heard over the second interspace; this murmur was heard when the patient was discharged from the hospital.

The fourth case,[1] a patient under the care of Dr. Andrew Clark in the London Hospital, a female æt. 20, who was admitted into the hospital March 2nd, 1868. She stated that this was her first attack of rheumatic fever; that she was well three days before admission. Two days before admission she was taken with pain in the left ankle, which extended the following day into the opposite ankle. She was admitted into the hospital two days after the rheumatic symptoms first appeared, that is, on the third day of seizure, and there was, at that time, proof of the heart being involved. A systolic mitral bruit was heard at the apex of the heart, which continued and was distinctly heard on the twenty-third day of admission.

The fifth case, a female æt. 18 (*vide* Case 7, 'Guy's Hospital Reports,' vol. xii, 3rd series, p. 525), was admitted into Guy's Hospital December 20th. The rheumatic process commenced five days before entering the hospital. On the day of admission a bruit de cuir-neuf was heard over the base of the heart, and subsequently a decided systolic bruit became developed.

In a sixth case, a male æt. 23 (*vide* Case 3 in the same number of 'Guy's Hospital Reports,' p. 515) was admitted into Guy's Hospital November 3rd. It was his first attack of rheumatic fever. On the seventh day of the rheumatic process there was a systolic mitral bruit at the apex of the heart, which persisted and remained when the patient was discharged from the hospital.

Referring back to these six cases, we find that two patients were admitted two days after the rheumatic process commenced, one three days afterwards, one four days, another five days, and lastly one seven days after the rheumatic process set in. In all these six cases there were physical signs showing that the heart was involved.

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[1] The record of this case does not appear to have been included in this paper.—[Ed.]

Of twenty-two patients[1] suffering from a first attack of rheumatic fever, thirteen came into the hospital during the first week of the disease. In one patient the rheumatic fever had commenced five days before admission into the hospital, in another six days before admission, in a third three days before admission, and in the remaining cases the disease had been going on five, six, five, four, five, six, one, six, three, and five days before the patient entered the hospital. The remaining nine of the twenty-two patients had been ill longer than a week when they were admitted into the hospital.

Every one of these thirteen patients on the day of admission into the hospital was found to have organic disease of the heart, and the heart disease had commenced before the patients entered the hospital. In these cases, therefore, the heart must have become diseased before the second, third, fourth, or fifth day of the rheumatic fever; in all these cases the heart was diseased before the patient had suffered from rheumatic fever a week.

It appears to us, therefore, that these cases prove that in rheumatic fever the heart is very liable to become diseased during the early stage of that disease, and that it very frequently does become diseased during the first week of the malady.

Further, two cases showed that the heart had become diseased as early as the third day, and in one case that it had become diseased two days after the rheumatic process had set in.

Having shown that the heart frequently becomes diseased during the first week of the rheumatic fever, we now proceed to inquire if it is common for it to become diseased during the second, third, or subsequent weeks of rheumatism.

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[1] Many of these are our own cases reported in vols. xi and xii of the 'Guy's Hospital Reports,' 3rd series. Others are taken from Dr. Garrod's cases (*vide* Royal Med.-Chir. Soc. 'Trans.,' vol. xxxviii). These twenty-two patients have been selected simply because they were suffering from a primary attack of rheumatic fever, and the heart was diseased on their admission into the hospital.

To prove that a patient who has escaped heart affection in the first week of rheumatic fever is not liable under any circumstances to suffer from heart disease in the second or subsequent weeks would require a very large experience, and such an opinion ought to be supported by a very large number of cases. A few cases may, however, be sufficient to show that there is no great tendency for the heart to become diseased during the second and later weeks of rheumatic fever.

We have particulars of twelve cases of well-marked rheumatic fever, seven of which occurred in the London, and five in Guy's Hospital. In not one of these cases was there any organic disease of the heart when the patients entered the hospital; eight of these twelve cases entered the hospital during the first week of the rheumatic fever. Four of them entered the hospital five days after the rheumatic symptoms first appeared, two six days after, one four days, and one three days after. The remaining four of the twelve cases did not enter the hospital until after the rheumatic fever had been going on eight, eight, twelve, and twenty-one days respectively.

These eight patients were placed under the mint water treatment; the rheumatic fever was, therefore, allowed to run its natural course.

All these patients were admitted into the hospital during the first week of the fever free from organic disease of the heart, and while they were under treatment during the second and subsequent weeks of the seizure no heart disease came on. In not one instance did the patient escape heart disease during the first week of the rheumatic fever and contract such disease during the second or later weeks of the fever.

If the heart was not diseased in the first week it did not become diseased in the second or in the later weeks of the rheumatic fever.

It will be instructive now to inquire if what we have stated respecting the heart becoming diseased in the first and not in the second week of rheumatic fever in any way explains why the heart did not become diseased when patients were in the hospital and treated by alkalies, lemon-juice, blisters, or by the expectant plan.

The explanation would, according to what we have shown, appear to be that patients suffering from rheumatic fever are not, as a rule, admitted into the hospital until the period when the heart is liable to become diseased has passed over. Experience teaches that the heart becomes diseased at the very outset of the rheumatic fever, before the patients enter the hospital; and if the patients pass the first few days of the rheumatic fever without the heart becoming involved, then they do not contract heart disease during the later part of the rheumatic attack.

Some patients treated by mint water entered the hospital as early as the third, fourth, and fifth day of the rheumatic fever without any heart disease, and the heart did not become diseased while the patients were under treatment.

Some patients treated by blisters or by alkalies entered the hospital as early as the third, fourth, and fifth day without any heart disease, and the heart did not become diseased while the patients were under treatment. The same thing occurred, therefore, when the disease was allowed to run its natural course as occurred when the disease was treated by blisters or alkalies.

In our cases treated by mint water the rheumatic fever commenced, on an average, eight days before the patients entered the hospital.

In Dr. Herbert Davies' cases treated by blisters, the rheumatic fever commenced, on an average, 7·9 days before the patients entered the hospital.

In Dr. Garrod's cases treated by alkali, the rheumatic fever commenced, on an average, about eight days before the patients entered the hospital.

So that Dr. Davies', Dr. Garrod's, and our own cases entered the hospital, on an average, about eight days after the rheumatic fever had commenced—that is, in the early part of the second week of the rheumatic fever. And the same thing was observed in Dr. Davies', Dr. Garrod's, and in our own cases, that if the patient's heart had not become diseased in the first week of the rheumatic fever—that is, before coming into the hospital—it was very rare for it to become diseased during the second or later weeks of the rheumatic fever while the patients were under treatment in the hospital.

However much influence we may consider that the blisters or alkalies had in preventing the heart becoming diseased, this fallacy was avoided in our own cases, for no drugs having any influence were used; and as the same result followed when drugs were not given as when they were given, it is difficult indeed to believe that the drugs were the means of preventing the heart becoming diseased.

We therefore respectfully submit that the reason why the heart did not become diseased when patients were treated by blisters or by alkalies was that the patients were not placed under the treatment until the period when the heart was liable to become diseased had passed over. For there is evidence tending to show that it is part of the natural course of rheumatic fever for the heart to become diseased during the first few days of the fever, and if it does not then become diseased it rarely does so in the second, third, or later weeks of the rheumatic fever.

We would here venture to observe that in acute rheumatism care is requisite in determining during the first week whether the heart is really diseased or not. Some physicians have considered that an unduly marked apex-beat, or an excited or an irregular action of the heart, indicated in rheumatic fever that the heart was becoming diseased. In some cases there may be no auscultatory signs to show that the pericardium is becoming involved, but there may be great præcordial pain, and sometimes pain between the shoulders, and these symptoms precede the auscultatory signs some hours or a day or two.

In the commencement of the heart disease there may be no loud bruit, but merely a prolonged first sound, or a slightly marked new-leather-creaking bruit over the base of the heart. Both these sounds may be very easily overlooked, and yet experience shows that such a prolonged first sound, not unfrequently, in the course of a few days, becomes a well-marked mitral bruit, and the new-leather-creaking bruit becomes a well-marked to-and-fro pericardial bruit. It also occasionally happens that the first sound is prolonged at the apex, and continues so until the patient is almost, if not actually, convalescent, and then this prolonged sound becomes a decided mitral murmur.

We may now briefly sum up the various conclusions arrived at in this paper as follows :

1. That rheumatic fever uncomplicated with any very severe heart affection tends to run its course in nineteen days, calculating from the time the rheumatic symptoms first set in to their termination.

2. That the average duration of the acute symptoms while patients were in the hospital and free from very severe heart complications was nine days.

3. That in nearly all the cases referred to in this paper there was some modification of the heart's sounds during some period of the rheumatic attack.

4. That severe pericarditis complicated with pneumonia may subside and leave the heart healthy, so far as we can test by our present means, without the aid of any drugs.

5. That when the heart was healthy on admission into the hospital, it was very rare for it to become organically diseased while the patients were under treatment by mint water, or, in other words, when the rheumatic fever was allowed to run its natural course.

6. That the evidence before the profession shows that the heart very rarely became diseased while the patients were under treatment (in the hospitals) ; and that this was the case when the patients were treated by alkalies, lemon-juice, or by blisters to the joints.

7. That there is not sufficient evidence before the profession to prove that any of the advocated remedies have power to prevent the heart becoming diseased.

8. That in concluding that the treatment has prevented the heart becoming diseased, we have overlooked the fact that there might be no tendency at the time the patients were under treatment for the heart to become diseased.

9. That in rheumatic fever the tendency is for the heart to become diseased during the first few days of the fever, and should it escape the early days of the disease, there is each day a lessening tendency to its implication : hence our cases would appear to show that if at the end of the first week of

the rheumatic fever the heart is free from disease, there is little or no tendency for it to become diseased during the later weeks, provided patients are treated by rest and regulated diet.

10. That the reason why the heart did not become diseased when rheumatic fever was treated by alkalies, or blisters to the joints, is to be attributed not to the influence of the drugs, but to the natural course of the disease. For the patients did not come under treatment until the rheumatic fever had been going on some days, and until the period when the heart was most liable to become diseased had passed over.

We ought not to conclude the paper without expressing our convictions, that hitherto the investigation into the therapeutics of the rheumatic process have been rendered all but valueless by the deficiency in preliminary data. There are as yet no reliable facts in the pathology of the process: we are ignorant of the essential state of which the rheumatic symptoms are an expression.

At present, therefore, as regards treatment, our cases seem to show that we are limited to a careful regimen of the patient. Rest, mechanical and physiological. Rest in the very outset of the disease. We ought not to wait until the rheumatic process has become well developed in the joints. For it appears to us that the heart becomes involved simultaneously with the joints, and by rest we hope to quiet the heart's action, and so prevent it becoming diseased.

To regulate the temperature. To moderate excessive skin function by sponging the surface of the body with tepid water. To allay pain by placing the patient in an easy position, and sometimes by opiates. To sustain the organic nerve-power by light diet, and occasionally by small doses of alcohol. To procure rest by the simplest means, especially avoiding such movements of the body as may excite the circulation. In fine, to place the patient in a physiological state of mean rest, if it may be so expressed, of the nervous, the circulatory, the muscular, and the digestive systems.

To do this fully will tax often all our energies, and we should not be just to ourselves if we did not here say that to attain this has been the object of our greatest care. It required often more consideration than was requisite for prescribing any supposed appropriate drug treatment. We are therefore, at present, advocates of the exactest treatment of the patient under acute rheumatism, though we may doubt the value of so-called specific drugs.

SECTION VI.

DISEASES OF THE SKIN.

ON A

CERTAIN AFFECTION OF THE SKIN,

VITILIGOIDEA α. PLANA, β. TUBEROSA.[1]

THE object of this communication is to call attention to a somewhat rare disease of the skin, which, so far as our observations extend, presents itself under two forms: namely, either as tubercles, varying from the size of a pin's head to that of a large pea, isolated or confluent; or, secondly, as yellowish patches of irregular outline, slightly elevated, and with but little hardness. Either of these forms may occur separately, or the two may be combined in the same individual. Under the latter circumstances we are able to trace the connection of the two through an intermediate series of gradations, which clearly demonstrate their essential relations.

It is doubtful whether this disease has been hitherto described. The only account which at all corresponds to it is that given by Willan, of vitiligo. He defines vitiligo to consist of "white, shining, smooth *tubercles* arising in the skin, about the ears, neck, and face, terminating without suppuration." Bateman adds, "This disease is somewhat rare, and perhaps but little known." The plate he gives of it is very unlike the appearances presented by the cases we have seen, yet the further description given by him would, to a certain extent, apply to them. "It is characterised," he says, "by the appearance of smooth, white, shining tubercles, which rise on the skin, sometimes in particular parts, as about the ears, neck, and face, and sometimes over nearly the whole body, intermixed with shining papulæ. They vary much in their course and progress; in some cases they reach their full size in the space of a week (attaining the magnitude of a large wart), and then begin to subside, becoming flattened to the level of the cuticle in about ten days. In other instances they advance less rapidly, and the elevation which they acquire is less considerable; in fact, they are less distinctly tubercular. But in these cases they are more permanent; and as they gradually subside to the level of the surface, they creep along in one direction,—as, for example, across the face, chequering the whole superficies with a veal-skin appearance. All the hairs drop out where the disease passes, and never sprout again; a smooth, shining surface, as if polished, being left, and the morbid whiteness remaining through life. The eruption never goes on to ulceration." We have extracted the whole description given by this author of vitiligo, that our readers may judge how near it applies to the cases we have to record. As many particulars are named in it which were not present in our cases, and also many are wanting which we have observed, there may be a doubt whether it is here applicable. The two forms of the affection are indicated, and perhaps the want of exact correspondence may be attributable to the want of a sufficiently large number of cases from which to frame a more accurate general description. Believing it to be probable that Willan would have included the cases here recorded under Vitiligo, or an allied affection, we have named them accordingly, distinguishing the two varieties by the terms Vitiligoidea tuberosa, and Vitiligoidea plana. We would note here that authors have not generally used the term Vitiligo as Willan and Bateman used it. Alibert limits it to a simple loss of pigment, without alteration of texture, and in this he is generally followed. Neither Alibert nor Rayer gives any description which would apply to the cases we have to record. The "keloide" affection of these authors is altogether of a different character.[1] It generally exists as a single tumour, arising either spontaneously or upon a cicatrix. Its course is remarkably slow, and leads subsequently to contraction and seaming of the skin.

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[1] By Thomas Addison, M.D., and William Gull, M.D. Reprinted from the 'Guy's Hospital Reports,' vol. vii, 1851, p. 625.

The above appears in the 'Guy's Hospital Reports' under the joint names of Dr. Addison and Dr. Gull; it appears also in Dr. Addison's collected works, edited by Dr. Wilks. The following note with regard to this paper was found in Sir William's handwriting after his death:—"This paper I wrote entirely, and coined the generic and specific designation, and pointed out its clinical relation to liver affection. When the paper was finished I asked the favour of Dr. Addison to let his name appear with mine at the head of the communication, which he did. My request was on the ground of gaining authority for what I had written, and I was then working with Addison, who saw the cases with me."—[ED.]

The following is an outline of the history of the cases which we have observed.

Several years ago, a young woman, æt. 24, was admitted into the hospital with a peculiar eruption, extending across the nose, and slightly affecting both cheeks. It consisted of shining tubercles, varying from the size of the smallest papule to that of ordinary acne. They were of a lightish colour, with here and there superficial capillary veins meandering over them, giving them a faint rose tint. The changes they underwent were very slow; whilst some advanced, others subsided. The further course of the case was not ascertained.

The Model[2] 2733$^{\text{1st}}$ presents an accurate copy of the appearances.

It was not until the winter of 1848 that our attention was again drawn to the subject, when the following case occurred: —Mrs. B—, æt. 42, of fair complexion and blue eyes, married, mother of eleven children, had been the subject of jaundice for two years, with much pain about the right hypochondria. After the jaundice had lasted fourteen months a change began in the integument, about the eyelids, and in the palms of the hands and flexures of the fingers. The skin was at this time of a lemon tint. The affection of the eyelids consists of *patches of a light opaque colour, with the surface and edges slightly raised,* extending from the middle of the upper lid inwards around the inner canthus, and then outwards along the lower lid to nearly the same extent. There is a small isolated patch at the outer canthus. The disease affects both eyes equally and symmetrically, with the exception of two spots in the right lower lid, about the size of a hemp-seed, more elevated than the rest. The cuticle over the affected parts is healthy. There is no appreciable induration. The patches are more sensitive than the surrounding parts. The capillaries of the cheeks are slightly tortuous. The palms of the hands are of an olive-brown; along the ridges on either side of the flexures, both of the palms and fingers, there is the same opaque, yellowish discoloration. The appearance is much as if the cuticle were thickened, and the disease confined to it; but, on a complete investigation, it is evident that here, as on the face, it is healthy, and that the morbid change is seated in the cutis, which is rather thickened, altered in colour, and has increased sensibility. The disease remained stationary until death, at the end of four years from the beginning of the jaundice. Towards the end the colour of the general surface deepened to a mahogany-brown. No affection of the skin, similar to that described on the face and hands, appeared elsewhere.

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[1] Keloide "c'est une excroissance faite aux depens du tissu cellulaire de la peau d'une configuration tantôt oblongue et cylindracée, tantôt ovale ou ronde, et bombée d'une couleur rose pale, dure et renitente au toucher, profondement adherente et comme incrustée dans le tegument offrant parfois à sa surface une multitude de petites veines injectées, imitant assez bien la forme d'une cicatrice qui succederait à une forte brulure poussant d'ordinaire vers ses bords des prolongemens bifurqués, qui ont quelque ressemblance avec les pieds d'une tortue ou les pattes d'un crâbe, phénomène constant qui justifie complètement la denomination qui a été imposée par M. Alibert à cette tumeur veritablement extraordinaire."—RAYER, 'Maladies de la Peau.'

[2] The models referred to throughout this paper are in the museum of Guy's Hospital.—[ED.]

The Models 2733^{2nd} and 2733^{3rd}, exhibit the appearances presented by this case.

On the 18th August, 1848, a patient was admitted into Guy's Hospital, under the care of Dr. Hughes, for diabetes. The following is an outline of his history at the time:—John S—, æt. 27, of middle stature; by occupation a tailor, residing near Kingsbridge, in Devonshire. About six months before admission he began to pass an unusual quantity of water, feeling at the same time weak and feverish, with a dry, harsh skin. On admission he presented the ordinary symptoms of diabetes; he voided four pints and a half of urine daily, sp. gr. 1050. The treatment pursued was various, but without any obvious improvement. On the 25th January of the following year (1849) the quantity of urine was seven pints and a half, sp. gr. 1042. At this time an eruption somewhat suddenly appeared on the arms, at first apparently of a lichenous character. In the course of ten days it had extended over the arms, legs, and trunk, both anteriorly and posteriorly, also over the face and into the hair; it consisted of *scattered tubercles of various sizes*, some being as large as a small pea, together with shining, colourless papules. They were most numerous on the outside and back of the forearm, and especially about the elbows and knees, where they were confluent. Along the inner side of the arms and thighs they were more sparingly present, and entirely absent from the flexures of the larger joints. Besides the compound character produced by the confluence of two or three tubercles, many of the single ones had also a compound character, or appeared to have such, as shown by the prominent whitish nodules upon them. Some looked as if they were beginning to suppurate, and many were not unlike the ordinary molluscum, but when incised with a lancet they were found to consist of firm tissue, which on pressure gave out no fluid save blood. They were of a yellowish colour, mottled with a deepish rose tint, and with small capillary veins here and there ramifying over them. They were accompanied with a moderate degree of irritation, hence the apices of many were rubbed and inflamed. The nature of the eruption gave rise at the time to much discussion. On its first appearance some suspected it to have a secondary venereal affection; but there was nothing in the case, nor indeed in the character of the eruption, when carefully examined, to support this view. The only cutaneous affection with which we could associate it, was that of the woman, whose case we have given above, where the tubercles had occurred in the face only. The eruption continued almost stationary from the end of January to the beginning of March, when many of the tubercles began to subside, leaving no obvious change in the texture of the skin. At the end of March the patient left the hospital, and the further course of the case was not ascertained. The appearances presented by the eruption in this case are well shown in Model 2733^6th.

Up to this time we had, therefore, these three cases of anomalous affection of the skin, without being able to do more than suspect a relation between the first and the third. Some further light was thrown upon the subject by the following case.

Eliza P—, æt. 33, of middle stature, moderately well nourished; mother of six children; catamenia regular. Her present illness began in 1848; she attributes it to fright, and to a blow received in the left groin whilst attempting to separate two men who were fighting. Two days after this she became jaundiced, and had from time to time severe paroxysmal pains about the hypochondria, lasting for a day or two, the liver being also enlarged and tender. Four months after the commencement of the jaundice, (August 4th, 1848) she was admitted into the hospital under the care of Dr. Hughes. She remained in until the 26th of September, and left much in the same state she was in when admitted. There was at this time nothing complained of beyond the itching and irritation of the skin common in jaundice. The present affection began after the jaundice had continued fourteen months, when she again came under the care of Dr. Hughes. It first appeared in the hands, spreading across the flexures of the joints of the fingers and palms. Soon afterwards a yellowish patch of discoloration began near the inner canthus of the eyelid, and then a precisely symmetrical one at the same part on the opposite eyelid. These patches are very slightly raised, and not obviously indurated; they have extended very slowly. In the early part of the year 1850, two models, 2733^4th, 2733^5th, were made of the case. At this time the patches on the face existed as above described. Along the ridges bounding the flexures in the palm and about the joints of the fingers, there were yellowish, opaque, irregular, and somewhat raised lines. About the thumb, first joints of the fingers, and inner and interior parts of the wrists, there is a gradual transition to a tubercular prominence of the affected parts, and some distinct tubercles exist on the elbow and knee. The diseased parts are tender, so as to give her pain in using a knife to cut bread. The whole surface of the body is of a dull lemon tint. Various means were employed without avail, the disease showing a tendency to progress slowly. Through the kindness of Mr. Startin, under whose care the patient now is, we have been able to observe it up to the present time. The jaundice still remains, occasionally deepened by the exacerbation of the hepatic symptoms. The skin is of a dull lemon hue. During the last seven months the affection has become more tubercular, especially about the back of the joints of the fingers of the right hand. The patch of confluent tubercles on the elbow has much increased since the model was taken. Both elbows are similarly affected. There are also tubercles on the right knee, on the superior surface of the great toe, and on both ears. On the hands the gradations from the plane to the tubercular variety are well marked, and the essential relations of the two forms demonstrable. This case has been of the greatest value in enabling us to connect together the cases which had previously occurred. The tubercles about the ears, elbows, joints of the fingers, &c., are of the same character they were in S—'s case (p. 554). They are firm, rather irregular on the surface; have much the appearance, at first sight, of small compound follicles, but on closer inspection are proved to depend upon a change in the cutis. On the surface small venous capillaries may be here and there seen, producing a mottled appearance. In the hands we pass insensibly from the tubercles on the back of the joints to the state described in Mrs. B—'s case (p. 553), namely, the slightly raised, opaque, yellowish lines about the flexures of the palms and fingers. The further identity of the disease in the two cases is shown by the presence of similar patches about the eyelids in both.

Mrs. J—, æt. 43, of spare frame, and below the middle stature, married; mother of two children, and in good health until about eight years ago, when her catamenia ceased, probably from fright. After their cessation she was never well, had pains about the right side and through the shoulders; and for several years past—indeed, nearly ever since the commencement of her ailment—has been jaundiced. She was constitutionally of a dark complexion;

this has now become a deep olive brown. During the last five years there has been a gradual change in the integument of the eyelids, giving her a strange expression. This affection of the skin began in the upper lid of the left eye, and extended round by the inner cauthus to the lower lid. A similar affection then commenced in the right eyelid, and the appearances now presented by the two are remarkably symmetrical. The surface of the affected parts is slightly raised, and the edge defined. The colour is a light opaque yellow—"coloration feuille morte," with a mottling of the faintest rose tint, with a small meandering vessel or two, especially on the patches, which are recent and extending. On passing the finger over the surface there is a slight, yet but very slight, feeling of resistance. The older spots are the most raised. The cuticle is unaffected, and by slight tension of the skin, will be seen to pass unchanged from the normal to the diseased parts. The discoloured patches often smart, and, to use the patient's expression, "seem as if gathering;" they have also an increased sensibility.

It will be observed, that as the disease extended, it has run along the lids so as to avoid the Meibomian region, and that in the left eyelid are two sebaceous follicles, enlarged and filled with dark pigment cells; during the last two years a spot of black pigment has appeared on the mucous membrane of the lower lip. The whole course of the disease has been very slow, and its increase, by degrees, almost insensible. There is no affection of the skin of any other part of the body beyond the change in its colour above indicated. The liver is enlarged, and there is much tenderness about the left hypochondria. The urine contains bile, and the conjunctivæ are of a decided jaundiced tinge.

We have preferred thus recording the cases which have formed the source from which our knowledge of this affection is drawn, to giving any more abstract dissertation upon it; hoping thus that the experience of others may be the more easily compared with our own, for, doubtless, cases of the like kind have occurred to most; although, until attention is especially drawn to a subject, the individual importance of isolated cases is apt to be overlooked. The connection of this affection of the skin with hepatic derangement is obvious, and the exception which occurred in diabetes is of the more interest, inasmuch as modern pathology points to the liver as the faulty organ in this disease. In what way the defective action of the liver operates, can, perhaps, be no further explained at present, than by the general theory of disordered circulating fluids. It is a matter of experience, that various affections of the cutaneous surface, such as numbness, itching, lichen, urticaria, &c., are closely connected with jaundice, depending, probably, upon the direct action of the morbid fluids upon the cutaneous tissues.

The treatment of these cases has been hitherto unsuccessful. They have manifested an inveteracy equal to that of the morbid conditions of the liver, with which they seem to be associated. In the case of S—, many of the tubercles had slowly subsided before he left the hospital, but in the others there was no tendency to disappearance, especially in the patches about the eyelids. Some slight benefit seemed to follow the careful and repeated application of nitrate of silver, but when the disease is extensive this would hardly be practicable. Mrs. P— informs us that although none of the tubercles have disappeared, yet they are now rather less prominent than they were a year ago.

In the subsequent volume of the 'Guy's Hospital Reports'[1] the following note is added, giving details of the progress of Mrs. P—'s case, and a drawing of the hands illustrating the transition of the variety "plana" into the "tuberosa."—[Ed.]

Vitiligoidea, as we have shown, may occur under either form separately, and for some time it was difficult to associate the two conditions. In Mrs. P—'s case they occur together, and in the hands the plane passes insensibly into the tuberose form. When the eruption first appeared in the hands, it was limited to the flexures of the joints of the fingers, and of the palms, producing to the superficial observer an appearance not unlike psoriasis palmaris, but with this easily recognisable difference, that in vitiligoidea the cuticle is unaffected over the diseased parts. The morbid change gradually extended over the fingers, at first producing scarcely any elevation, as in the patches round the eye, and coincidently with this extension, raised tubercular masses formed on the knuckles. During the past year the disease has continued to progress, with the exception of the spots round the eyes. The skin of the palm is now becoming raised and tuberose, as on the back of the joints, and the hands are so tender that she is scarcely able to use them. Similar formations have taken place on the nates. The disease on the elbows and ears has advanced, as in the other parts. Jaundice remains; the nutrition is good.

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[1] 'Guy's Hospital Reports,' vol. viii, 1853, p. 149.

The nature of this remarkable affection is still obscure. It is important to note that during the last few months the tendons over the metacarpo-phalangeal articulations have become tuberose, having apparently undergone a change similar to that of the skin, although the integuments over them are unaffected.

ON THE

PARASITICAL VEGETABLE NATURE

OF

PITYRIASIS VERSICOLOR

(*Microsporon furfur*, ROBIN).[1]

In 1846, Eichstedt found a fungus growing amongst the epithelial scales in pityriasis versicolor. The discovery was confirmed during the following year, by Slyter, in a tract 'De Vegetabilibus organismi animalis parasitis ac de novo epiphyto in Pityriasi versicolori obvio.'

In this country, and in France, the subject still seems to remain in doubt. Robin, in his elaborate treatise 'Des Végéteaux Parasites qui croissent sur les animaux vivants,' details the observations of the above-named authors, but adds in a note, " Je n'ai pu verifier moi-même ces faits." Wilson, in an edition of his work on 'Diseases of the Skin,' so recent as 1851, says, "Dr. Gustav Simon places chloasma (*Pityriasis versicolor*) in his sixth group of diseases of the skin, which he entitles *Parasites,* considering this eruption as depending, like favus, sycosis, and alopecia circumscripta, upon the presence of a parasitical vegetable fungus. I do not agree with him in this opinion, and have failed to discover any vegetable organisms, although I have searched for them with care."

In an edition of Cazenave's lectures, published in 1853, there are the following remarks :—" Envisagé dans son ensemble le pityriasis est une affection complexe (*sic*) dont le caractère intime est exprimé par la double existence d'une secrétion anormale de la matière epidermique et d'une lésion de secrétion de la matière colorante."

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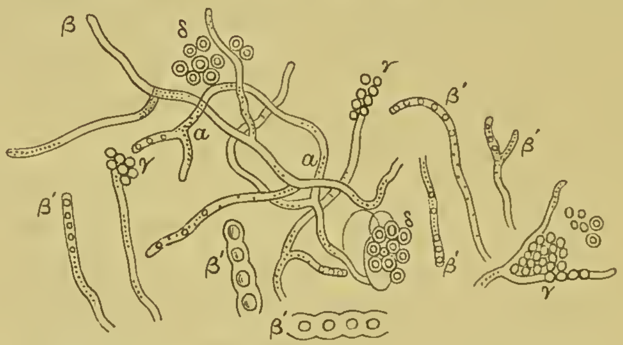
[1] Reprinted from 'Guy's Hospital Reports,' vol. ii, 1856, p. 191.

With the subject in this uncertain state, it will not be superfluous to record any observations which may bear upon it. In the several cases where I have looked for the fungus in pityriasis versicolor, I have always found its sporules amongst the scales of the epidermis; but until recently, I have failed to detect the ramifying branches of the mycelium itself. This failure I now know to have arisen from want of care in manipulation. The description of the fungus given by Robin[1] agrees with what I have observed, except the rarity and difficulty of making out the terminations of the filaments, which I have not found. It will be seen from the subjoined sketch, that the filaments sometimes end

Sketch of the Microsporon furfur.



α α Mycelium. The filaments $\frac{1}{8000}$ to $\frac{1}{10000}$ inch.
β Simple termination.
β' β'. Terminations containing sporules.
γ γ. Terminations as receptacles.
δ δ. Sporules free, $\frac{1}{4000}$ to $\frac{1}{5000}$ inch.

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[1] Trichomata (fila) in squamis epithelialibus sita, nunquam etiam earum marginem excedentia, multipliciter torta et inter se nexa ut raro fili finis cujusdam certo cognosci queat; simplicibus parallelis lineis terminata nunquam aut articulata aut in margine vincta nec contenti quid in eo apparet; passim in ramulos divisa. Sporidia rotunda binis adumbrantur lineis concentricis quarum interior spatium lucidum circumdat in acervulis agminata. (Robin, op. citat., p. 436.)

simply, at others, by a slight enlargement, as a sporangium, with the sporules in a linear series, and at others form a receptacle with the sporules more or less regularly disposed upon them.

To anyone who will be at the pains of investigating the structure here described, it will be unnecessary to submit the arguments for its vegetable nature. The character of the branching filaments, the mode of their fructification, and the form and structure of the cells (sporules) so produced, associate it with the fungi, and distinguish it from all, even the lowest forms of animal productions : yet there still prevails a contrary opinion.

Mr. Wilson, in discussing the vegetable nature of the epidermic growths in *Porrigo lupinosa*, makes use of the following reasoning :—" From analogy," he says, " the mode of development and growth of a cell must be the same, in whatever part of the body it is produced, and whatever special purpose it may have to perform ; and microscopical investigation establishes the existence of an identity of structure among them. The blood-cell, the mucus cell, the pus cell, the pigment cell, the epithelial or epidermal cell, for example, resemble each other closely (*sic*) in construction, and in some instances appear to be convertible the one into the other. The cells or corpuscles of favus possess a striking resemblance to pus cells, and excepting in their form, are closely allied to young epidermal cells, so that it would require no stretch of imagination to suppose the epidermal cell, altered in its actions by disease, capable of assuming the character of a pus cell ; or the latter, from a similar cause, passing into the likeness of a favus cell." After this apology for such a conversion, the author proceeds to detail his observations on the change of pus into the structures in question. The examinations which I have made of the favi in different stages, on many occasions, lead me, however, to an opposite conclusion. I could never trace any intermediate steps to support the theory offered by Mr. Wilson. The cellules, to which the name of sporules has been given, have all the physical and chemical properties of the sporules of the lower fungi, and the stages by which they develop filaments and fructify are readily traceable, which would certainly not be the case were they pus cells, or any other form of exudation cell. An argument like this adduced by Mr. Wilson against the vegetable nature of these productions, was, not many years ago, used by Professor Owen against the independent animal nature of the acephalocyst hydatid. "The knowledge," he says, "that we now possess of the primitive embryonic forms of all animals, and of all animal tissues, places us in a position to take a true view of the nature of an acephalocyst. It seems to be most truly designated as 'a gigantic organic cell,' not as a species of animal, even of the simplest kind. . . . . The primitive forms of all tissues are from cells, which grow by imbibition, and which develop their like from their nucleus of hyaline. All animal tissues result from the transformations of these cells. It is to such cells that the acephalocyst bears the closest analogies in physical, chemical, and vital properties;" yet, notwithstanding this connected chain of analogical reasoning, by so acute an observer, we are at this day in a condition to see its fallacy, and to assert, on the contrary, that physically, chemically, and vitally, there is no similarity (at least such as implies identity) between the texture of the hydatid cell and of those which constitute the embryonic forms of normal textures.

Every advance in knowledge points to a similar negation of the analogical arguments adduced by Mr. Wilson, and confirms the opinion of Continental pathologists, that the surfaces of the body are subject to true vegetable parasites, as they, and other textures, are to animal parasites. In any given case, therefore, it appears to be not a question of the vegetable nature of these formations, but whether they are accidental, and their presence determined by some prior morbid process, or whether they constitute by themselves a substantial and independent cause of disease. Dr. Hughes Bennett[1] maintains the opinion, that all such formations are secondary, and "always arise in living animals previously diseased." That a large proportion of them owe their existence to such circumstances is probable; as the *Sarcina ventriculi*—the fungi found in thrush; on pulmonary tubercle; on the pleura in pneumothorax; in saccharine urine; in the sordes and mucus of the mouth; in the discharges in chronic disease of the ear; and in the decomposing evacuations in cholera. On the other hand, there are others which cannot be so disposed of, and which appear to fix on previously healthy surfaces, and to be in themselves the essential cause of disease—as the *Tricophyton tonsurans* in porrigo scutulata, the *Microsporon furfur* in pityriasis versicolor, the *Microsporon Audonini* in one of the forms of porrigo decalvans, and the *Achorion Schönleinii* in porrigo lupinosa.

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[1] 'Transactions of the Royal Society of Edinburgh,' 1844, p. 291.

Though I can confirm the statement made by Bennett and Wilson as to the more frequent occurrence of this last in scrofulous subjects, yet I have seen it where the general health was unexceptionable.

In a practical point of view, the distinction between the two classes is of value, since, if we have once ascertained what affections are essentially local, our treatment directs itself accordingly, instead of leaving us to hull about in a sea of conjecture as to constitutional causes and predispositions which have no existence.

As little does it avail to attempt the cure of pityriasis, and the like affections, by constitutional means, as it would of scabies. As in the latter we seek to destroy the parasitic animal and its ova, so, in the former, the fungus and its sporules. A cachexia may be present in either case, requiring appropriate treatment; but, whether present or absent, the local disease gives the same therapeutical indications. It is not the object of these notes, however, to discuss the treatment of the vegetable diseases of the skin, but I may add that I have found the acidum aceticum diluted with four or six parts of water effectual in clearing the skin of pityriasis versicolor. The above remarks apply only to this variety of pityriasis. There are, it is well known, other forms which arise from an abnormal production of epithelium, in which no vegetable structure takes any part.

ON

FACTITIOUS URTICARIA.[1]

WILLAN describes a form of urticaria which shows itself when the skin is rubbed. "It may," he says, "be excited on any part of the body, in a few seconds, by strong friction or scratching, but the wheals presently subside again." It is included by this author in "urticaria evanida" because the wheals are transient, nor does he distinguish it from ordinary chronic urticaria, with wheals arising spontaneously. It is this distinction which the term "factitious" is intended to mark, for the malady thus named (when it amounts to a malady) is not associated with common urticaria, the skin having its natural appearance in the cases I have seen, unless rubbed by the dress or otherwise, or stretched by the use of the muscles in violent exertion. The patients were not liable to the eruption of any wheals but such as had this mechanical origin. Any form of wheal in these cases could be determined by the direction and extent of friction on the skin, and if with a blunt point any figure or letter was traced it quickly came into relief with great sharpness of outline.

The effect is greatest where the skin is well supplied with muscular fibre-cells, and hence on the volar surface of the thumb it is scarcely produced.

This susceptibility of the skin is common, in a greater or less degree, to all persons, and can be termed morbid only when extreme. In the first patient in whom I noticed it, it was the source of great inconvenience, for if the skin was handled roughly, as in wiping the face with a towel or in pulling on the socks, it would quickly become swollen and stiff with wheals. This susceptibility may be hereditary. It was so in the case of the gentleman who afforded his arm for the sketch. His father's skin was equally irritable. In four other cases which I have seen, this tendency in the skin came on gradually, without any assignable cause. In one only was there any recognisable disturbance of the general health, and that was in a youth of fifteen, who was liable to attacks of spasmodic asthma, which seemed to be associated with the state of the skin.

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[1] Reprinted from the 'Guy's Hospital Reports,' vol. v, 1859, p. 316.

When it was found that a wheal of any shape could be made upon the healthy skin, it suggested a more minute inquiry into the nature of a wheal, and the conclusion arrived at was that wheals are principally due to contraction of the muscular tissue of the skin. If a line be traced with slight force on a skin which is prone to this form of contraction, the first noticeable change is a wrinkling of the surface, as in "cutis anserina." In forty seconds there is a slightly raised red line; in sixty seconds the line is palpably raised and hard; in ninety seconds there is an obvious wheal, which becomes fully developed in three minutes. If a large space be rubbed there is a sensation of tightness and stiffness, as if the part were hidebound. If two points be marked on the skin previous to the friction, they are found nearer together after the wheal has risen. With the rising of the wheal, which is white and firm, there is an accompanying areola of capillary hyperæmia, which, after some minutes (fifteen or twenty-five), disappears, leaving the wheal for a longer time persistent.

If, as here suggested, a wheal be due to muscular contraction, we should, *a priori*, expect its formation and duration to be modified by whatever influences the activity of the involuntary muscular fibre, and such is the result of experiment. After dropping chloroform on the skin, however susceptible it might have been before, no wheal could be brought out by friction, and when chloroform was applied to a wheal already risen it quickly reduced it. When ice was applied to the part immediately after friction, a wheal did not rise. By stretching the skin the wheal could be obliterated, apparently by overcoming the contraction of the muscular tissue. These facts are elucidated by a collateral experiment. If the stomach and intestines of a cat or dog, immediately after death, be exposed, imperfect wheals (but essentially wheals) may be formed upon them by passing a point sharply over their surface. These are plainly the result of muscular contraction.

When we consider the rapidity with which the eruption forms, its noticeable gradations through cutis anserina, the hardness and sharpness of its outline, the conditions which modify it, and the parts of the surface where it is most readily excited, and its non-occurrence on others, the conclusion above stated seems irrefragable.

How a wheal is excited in ordinary urticaria, whether directly by the circulation of some irritating substance through the cutaneous tissues, or indirectly by reflection through the vaso-motor nerves, or indifferently by both modes, is not proved. Admitting that the blood is the more common channel, there are still many clinical facts, as well as the proofs given above, which show that it is not the exclusive one. It is well known that exciting or depressing emotions will favour the eruption, and sometimes it seems to be caused by merely thinking of it. We are so much in the habit of viewing these phenomena as evidence of the elimination of a *materies morbi*, as an "effort of nature" to throw off what is offensive, that we are prone to overlook all other modes of action.

SECTION VII.

MISCELLANEOUS PAPERS.

ON THE

EFFECTS OF ETHER ON THE DIFFERENT CLASSES OF ANIMALS.[1]

If a mammal or bird be made to inspire atmospheric air strongly impregnated with ether vapour, in from sixty to ninety seconds its muscular power is lost; it becomes totally insensible to pain; its respiration becomes slow and irregular in rhythm; the venous blood is of a vermilion colour; the heart beats with great rapidity; its rhythm is irregular, and its force diminished. If the experiment be made with a frog, the effect is produced in nearly the same time; considering, therefore, the slowness of its respiratory movements, the mixed nature of its circulating currents, and the lowness of its temperature, it becomes more rapidly affected than a mammal or a bird. The state of etherisation, when induced, is also more perfect and lasting than in warm-blooded animals, the respiratory movements in frogs being often arrested for six or seven minutes. Alcohol vapour produces similar effects; they are slower in their accession and more lasting in their effects, often fatal.

An increased flow of saliva is one of the ordinary effects of the inhalation of ether; this was observed in man, in cats, mice, and birds. The irregularity of the muscular movements which come on amongst the early effects of etherisation do not seem to depend so much upon want of muscular power as upon a loss of muscular sense—that is, the power of appreciating the force of the muscular contraction, and the exact locality of the limb.

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[1] A paper read before the South London Medical Society, April 15th, 1847. Reprinted from the 'London Medical Gazette,' vol. xxxix, p. 777. (The paper is reproduced as it is reported. Its historical value is so great that it could not be entirely omitted. The complete paper, if it was ever written, has been lost.—Ed.)

The phenomena produced by the inhalation of ether vapour are allied to those which result from alcoholic drunkenness; the former are more transitory, and more speedily induced. If drunkenness either by ether or alcohol is extreme, it will terminate in asphyxia. Some of the phenomena of ether drunkenness are induced by concussion. In both states the surface is often cold, pulse irregular and frequent, respiration irregular in rhythm and force; no recollection of occurrences during the stage of concussion or etherisation; memory of events long past wonderfully recalled both by concussion and etherisation. Ether in some persons produces extreme faintness and sickness, with trembling and paleness; concussion does the same. In concussion, as in etherisation, patients are occasionally violent, swearing and manifesting the phenomena of drunkenness. It has been considered a remarkable fact, that, by the inhalation of ether, common sensation should be lost, whilst the senses of hearing and seeing are so little impaired. Such a state is occasionally observed in nervous exhaustion alone; two classes of such cases might be enumerated,—bilious disorders, and after venereal excesses. In such cases the gait and other muscular movements may be awkward, arising from this diminution of sensation only, and not from muscular weakness.

The phenomena arising during the inhalation of ether are not at all dependent upon super-carbonisation of the blood. Frogs are rapidly affected by ether vapour, whilst they may be kept for hours in hydrogen and nitrogen without injury. Pure ether vapour killed a bird past recovery in twenty-five seconds. Ether probably permeates every tissue, but acts most upon the nervous, on account of its physical constitution, the grey substance of the brain containing 4·5 per cent., the medullary substance 14·5 per cent. of fatty matter. The irritability of muscular fibre in frogs is not evidently diminished by ether. Ether probably produces its effects by direct action on the tissues, independently of the quantity of blood in the part. The first effect seems to be an increase of the function, or stimulation; whether this is followed by an increased supply of blood, according to the prevalent law of nutrition, it is not easy to say. If the action of the ether vapour be continued, a loss of function follows, or what is termed its *sedative* effect; this may be the cause of death, or the function of respiration may in the higher animals be depressed for so long a time that asphyxia may come on. Congestion is not a direct result of the action of ether vapour on the brain, but, as in ordinary alcoholic drunkenness, so in ether drunkenness, asphyxia may be induced, and death follow.

Effects of ether on the blood.—The rigor mortis is well marked in animals killed by ether vapour. The blood has a strong odour of ether. After the inhalation of ether it may be detected in the breath for thirty-six or forty-eight hours. Blood drawn from a vein whilst the animal is fully under the influence of ether has a vermilion colour, and coagulates firmly. The formation of the globules is unchanged.

All other things being equal, it is probable that the fitness of a patient for the inhalation of ether, if that should be desirable, would be determined with more certainty by observations on the nervous system, vascular plethora by no means so far contra-indicating its use as a feeble nervous system. This opinion is based on the known effects of ether on man and the lower animals, especially the cold-blooded. Frogs, when fully under the effects of ether, are yet strongly affected by a single galvanic current, as may be proved by laying the animal so affected on a glass plate, and placing a shilling under the lower part of the spine, and a plate of zinc under the head, and connecting them with a copper wire.

Equal parts of ether vapour and oxygen gas produce a compound as rapidly fatal as ether vapour only. If an animal be etherised and then made to respire oxygen, it does not recover more speedily than if it merely respired atmospheric air. In many cases the respiration of oxygen after ether retards recovery, and in some is rapidly fatal, death following at once on the respiration of oxygen. These results were proved in birds, which breathe oxygen for several minutes without injury. Oxygen, therefore, is not an antidote to the ether vapour. Nitrous oxide gave results similar to those of oxygen.

It is an error to suppose that the action of ether is necessarily allied to asphyxia.

Most of the above statements were verified at the meeting by experiments on animals. The author ended his communication by the following queries upon the surgical employment of ether:

1st. Is it useful to abolish pain during a surgical operation?

2nd. Can this be done safely by ether?

3rd. Does the presence of ether in the blood modify the healing process?

In answer to Dr. Munk, the author stated that, if immersed in pure oxygen gas for a short time, the animal would be merely excited, no other effect being produced.

During the discussion which followed, Mr. Benjamin Travers, jun., remarked that he believed ether to be a poisonous and dangerous remedy, attended with the greatest risk, and requiring the most profound caution in its use.

Mr. Bransby Cooper, in reference to Dr. Gull's question "whether it was right in operations to alleviate or prevent pain, provided it could be done with perfect safety," remarked that pain was a premonitory condition, no doubt fitting parts the subject of lesions to reparatory action, and therefore he should feel averse to the prevention of it. Dr. Gull's paper had more than ever convinced him that ether was a poison, and unless other experiments proved it harmless, he should give his decided opinion against its use.

Mr. Cock's Case—to divide the Tendons of the Flexors; Operation under the Influence of Chloroform.[1]

Sir,—The following incident, which occurred at Guy's Hospital yesterday, seems to me of sufficient importance to trouble you with its details.

A boy, æt. 11, was under the care of Mr. Cock for disease of the right knee-joint, and it was determined to divide the tendons of the flexors.

The boy was in good health, but his nervous system a little weakened from confinement to bed; his heart and lungs sound.

A small quantity of chloroform, not exceeding thirty drops, was put upon a cone of bibulous paper and placed over his mouth and nose. In less than a minute he was entirely insensible, the pupils became widely dilated and the pulse small and frequent. As the operation was being proceeded with his consciousness partly returned, and a few drops of the chloroform were put on a handkerchief and applied to the nose.

He was instantly affected, and to such a degree that there was the greatest apprehension of his never rallying; the pulse was very feeble, 56; the breathing so indistinct as scarcely to be distinguished; the face pale; lips congested; the symptoms of collapse extreme.

Ammonia was employed, and after about five minutes he took two or three deep inspirations; it was, however, more than fifteen minutes before he was so far himself as to be considered out of danger.

Subsequently a small quantity of brandy was administered. He complained of headache.

For a long time after he recovered his special senses and power of motion, general and perfect anæsthesia of the surface existed. This morning he is quite well.

I am, Sir,
Your obedient servant,
WM. W. GULL.

December 8th, 1847.

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[1] 'London Medical Gazette,' vol. xl, 1847, p. 1036. (This letter has been reprinted as it is of great historical interest, and shows the rarity of operations under anæsthetics less than fifty years ago.—Ed.)

CASES

OF

PHLEBITIS WITH PNEUMONIA AND PLEURISY

FROM

CHRONIC DISEASE OF THE EAR.[1]

Amongst the cases of inflammation of the dura mater connected with disease of the ear, recorded by Abercrombie, is one of a young lady æt. 15, where death occurred in an intermediate stage of the progress of the disease in the ear from suppurative pleuritis; but whether he recognised the chest affection to be the *direct* result of the chronic changes in the ear or not we are left to conjecture, since he makes no particular remarks upon it. Dr Watson has more especially directed attention to this complication of general phlebitis with disease of the ear; although the illustrations he has given are defective, from the actual condition of the ear and the veins not having been ascertained by post-mortem examination. Dr. Wilde, in his work on 'Aural Surgery,' when treating of the cerebral affections connected with disease of the ear, goes on to say, "These cerebral symptoms do not, however, in every case appear to be the immediate cause of death. I remember two cases in particular, and I suppose they are the types of many others of the same class, where the lungs became affected in the latter stage of the disease, and in which the thoracic affection seemed to be the immediate cause of dissolution." He also quotes a case laid before the Pathological Society (of Dublin) in 1840, of a boy æt. 16, who, together with the cerebral symptoms arising from disease of the ear, had, towards the end, distressing cough, and severe pain along the right side of the neck. After death there was inflammation of the internal jugular and superior vena cava, with fœtid pus in the pleura, as well as purulent deposit in the cerebellum.

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[1] Reprinted from the 'Medico-Chirurgical Transactions,' vol. xxxviii, 1855, p. 157.

The following cases, which I wish to lay before the Society, are further contributions to this part of pathology. In neither of them had the cerebrum or cerebellum become the seat of disease, but the caries of the mastoid cells had set up local phlebitis, extending down the jugular vein, and producing an affection of the lung, which was, in all, the immediate cause of the fatal termination.

The first case is more especially worthy of note, as there were no external signs in the ear itself to direct attention to it as the seat of the primary irritation.

The occurrence also of pneumothorax is rare, although the condition of the lung in these cases is such as frequently to bring the patient to the verge of such an accident.

Phlebitis of the jugular vein, lobular pneumonia, pleurisy and pneumothorax, from chronic diseases of the ear.

Thomas T—, æt. 21, was admitted into Guy's Hospital, under my care, December 2nd, 1854, and gave the following account of himself:

He says that, a week ago, he took cold, whilst working as an engineer, in an exposed and marshy situation at Waltham. This was followed by sore throat, and stiffness about the neck. He has had several shivering fits, which he calls ague, frequent vomiting, and pain in the head.

Symptoms:—skin hot and dry; pulse 100; tongue injected, with two broad streaks of yellowish fur towards the centre; conjunctiva icterode. Complains of severe pain in the head. Face anxious; no delirium; abdomen contracted; muscles rigid; bowels inactive; nausea and vomiting; urine high-coloured without deposits; free from albumen. Pulmonary and cardiac sounds in all respects normal. Ordered—

Haust. effervescens, 4tis horis.

December 3rd.—Frequent vomiting; severe frontal headache; febrile symptoms as yesterday; no delirium.

5th.—During the last twenty-four hours has had two severe rigors, followed by increase of the febrile symptoms; no sweating. On placing him up in bed, to re-examine the chest, he is observed to move the head stiffly and cautiously. On inquiry, he says he has tenderness over the mastoid process, and down the neck behind the ramus of the jaw on the right side, with deafness on the same side. The external auditory canal, and the surface of the membrana tympani, appear to be free from disease. He states that four years ago, when working in a coal-mine in Staffordshire, he received a blow on the right side of the head, which was followed by the deafness and discharge from the ear. The tenderness and slight fulness in the course of the jugular vein, and over the mastoid, indicate this as the seat of the irritation giving rise to his symptoms. Ordered—

Hirudines, vj, pone aurem dextram.
Mist. Magnesiæ c. Mag. Sulph.,
ad sedes liquidas.

6th.—Two rigors since last visit; pain in the head relieved; no incoherence, nor any degree of cerebral oppression. This morning began to complain of a painful stitch in the right side of the chest. A harsh pleuritic rub audible over the part. Pulmonary sounds otherwise normal. Ordered—

Hydrarg. Chloridi,
Opii, ana gr. ss.
6tis horis.
Rept. haust. effervescens.

7th.—The swelling behind the angle of the jaw, and in the region of the internal jugular behind the ramus of the jaw, more distinct. Deglutition painful. The vomiting has ceased. Passed a restless night. About midnight had a severe rigor, lasting about an hour, and followed by sweating for near the same time. Skin now hot and dry; pleuritic pain and rubbing on the right side remain. Slight cough, with expectoration of a small quantity of tenacious mucus tinged with blood. Pergat.

8th.—A severe rigor last night, after which, to use his expression, he "could not get his wind for an hour or two." Now breathes tranquilly. Lies on his back. On exposing the chest, the right side was observed to be almost immovable and rounded; preternaturally resonant on percussion. The loud and harsh pleuritic rub of yesterday gone, and nothing now audible but bright amphoric breathing over the whole side (from pneumothorax?). At the base posteriorly, near the spine, tubular breathing, with mucous crepitation. On the left side, under the mamma, slight pleuritic rubbing, and over the whole side puerile respiration. Tongue dry and brown; pulse 120; no delirium; frequent short cough; no dyspnœa.

The diagnosis was *pleurisy and lobular pneumonia, from phlebitis, with perforation of the pleura pulmonalis.*

9th.—General condition as yesterday. A rigor about midnight lasting three quarters of an hour, not followed by sweating. Respirations 36; no dyspnœa; pulse 120; countenance suffused; slight cough. Condition of chest as yesterday. Pergat.

10th.—Dyspnœa urgent; respirations 48; pulse 138. Dulness over the inferior part of the left chest. Fine crepitation, extending upwards to the scapular and axillary regions, and anteriorly as high as the second rib. The same tympanitic resonance, and amphoric breathing, on the right side.

11th.—Died at half-past one p.m.

Inspection of the body, twenty-five hours after death, by Dr. Wilks.—Body well nourished; muscles firm and well developed; brain healthy, with the exception of a slight superficial discoloration on the side of the right lobe of the cerebellum, from contact with the diseased dura mater. The brain substance had not undergone any change of texture. The right lateral sinus, at its commencement in the torcular Herophili, contained an adherent clot; further on in its course the contents of the sinus were fibrine, partly softened into a greyish puriform fluid, and pus. The internal jugular vein was filled with the same at the upper part, and obstructed by an adherent clot as far down as its junction with the subclavian.

Over the petrous portion of the temporal bone, and continuous with the descending portion of the lateral sinus, the dura mater was sloughy. Pus lay between it and the bone, and was infiltrated along the coats, and into the sheaths, of the jugular vein.

The right pleural cavity contained a large quantity of air. The lung was collapsed to the spine. On blowing into the trachea with a blowpipe, air escaped freely through two openings in the lower lobe. These communicated with two small cavities, one of the size of a filbert, the other smaller, formed by a slough of the pulmonary tissue, the result of lobular pneumonia. The contents were ash-coloured, puriform and fetid. The pleura was covered by a considerable quantity of recent fibrinous exudation. There was about a pint of turbid fluid in the pleural cavity.

The left lung was universally affected with acute recent pneumonia, in the stage of early red hepatisation. A small amount of plastic exudation upon the pleura. Heart and pericardium normal. Liver healthy. Spleen large, weight ten ounces avoirdupois; tissue soft. Kidneys healthy.

The left ear was free from disease. The external auditory canal of the right ear was healthy; the membrana tympani intact, and the malleolus *in situ* upon it. The other portions of the chain of bones were loosened from their connections. The lining membrane of the tympanum was thickened and vascular. There was caries of the posterior and superior wall, and of the adjacent mastoid cells, extending to the dura mater at two points; in one as it lies upon the superior part of the petrous portion, and in the other as it covers the floor of the lateral sinus.

Phlebitis, pneumonia, and pleurisy, from chronic disease of the ear.

John Robertson, æt. 52, a native of London, and living nearly all his life in the neighbourhood of Bermondsey, married, and of sober habits, applied to me as an out-patient at Guy's Hospital, July 17th, 1854, complaining of feeling generally unwell, with headache and loss of appetite. Two days previously he had had a slight rigor. The tongue was moist and furred white. The case was regarded as one of fever, and he was accordingly advised to come into the house. Not consenting to this, he was ordered a saline mixture of the citrate of potash. At his second visit, the following week, he was admitted into the hospital. His symptoms were of the same character as before, and were for the first few days still set down to the invasion of continued fever. Every second day he had a rigor, lasting about ten minutes. This was attributed to the influence of the locality in which he had been living. The headache, general oppression, malaise, and furred tongue continued. The pulse was 70; skin moist. No enlargement of the spleen.

From July 24th to 28th he took—

Inf. Serpentariæ, ℥iss.
Ammoniæ Carb., gr. iv, 6tis horis.
Sherry, ℥vj.

On the 28th, the attacks of shivering returning daily, he was ordered—

Quiniæ Disulph., gr. iij, in pil.,
4tis horis.

August 8th.—The rigors still return daily, lasting for ten minutes, not now followed by sweating. Pergat.

19th.—Rigors less marked, but no improvement in the other symptoms.

Hydrarg. Chloridi, gr. v, st.
Ol. Ricini, ʒvj, post horas sex.

23rd.—No rigor for last five days. The general symptoms continue, and their cause remains obscure; sleep disturbed.

26th.—Attention was at this time drawn to the left ear and the parts adjacent. It was observed that he had stiffness of the neck, and pain in rotating the head. He now stated that from childhood he had had, at times, a discharge of offensive fluid from the left ear, and that he was deaf on this side. Ordered—

Jul. Hydrarg. Bichloridi C., ʒij,
bis die.
Empl. Lyttæ nuchæ.

31st.—Shiverings more severe; face anxious; tongue loaded and brown; with the exception of slight delirium at night, no disturbance of the cerebral functions.

September 3rd.—Complains of great pain down the left side of the neck, and towards the occiput. The parts are very tender, and he cannot suffer the hand to be moved on the spine. The rigors return daily, and are very severe. Tongue dry and brown; breathing frequent, with slight cough; Bedsores beginning to form on the back.

Died September 11th, 1854, twenty-five days from the accession of his symptoms.

Examination thirty-six hours after death.—Purulent discharge from the left ear. Underneath the skin and superficial textures, suppuration extending from the jugular fossa downwards and backwards amongst the muscles. The adjacent parts of the temporal and occipital bones, down to the condyles, were necrotic, and of a dark colour. The internal jugular vein filled with a soft fibrinous coagulum and pus. The lateral sinus contained the same. The external surface of the dura mater, over the petrous portion of the temporal bone, and the walls of the lateral sinus, were dark coloured and much thickened. The coats of the internal jugular were also thickened, and involved in the suppuration amongst the muscles. With the exception of a slight discoloration on the left lobe of the cerebellum, unattended with exudation or change of texture, the brain and cerebellum were healthy. Right pleura contained a pint of purulent effusion, with patches of recent yellow exudation upon the surface of the lung. Both lungs presented the different stages of pneumonic consolidation, in some parts approaching to a state of gangrene. Heart and pericardium healthy. Abdominal viscera healthy. The mastoid cells were extensively destroyed by caries, and, as in the preceding case, the bone was perforated in two places, at the superior part of the petrous portion to the extent of a third of an inch in diameter, and to the same extent into the sulcus lateralis. The disease had apparently extended into the mastoid cells from posterior wall of the external meatus, which at its commencement was cribriform from ulceration.

Phlebitis, lobular pneumonia, pleurisy, &c., from chronic disease of the right ear.

George Lewis, æt. 23, was admitted into Guy's Hospital, July 22nd, 1854, under the care of Dr. Habershon. He is ill developed, with a hydrocephalic head, boyish look, and half-fatuous expression. He has been living in great poverty, and been much neglected. For a fortnight before admission he had been suffering with febrile symptoms and slight delirium. The case was regarded as one of continued fever. On admission he was very prostrate; tongue dry, lips parched; skin hot and perspiring; pulse frequent; evacuations of a dark colour; urine scanty, high coloured, and containing an abundance of triple phosphate. A considerable discharge of thin, bloody pus from the right ear; pain at the back of the neck, much increased by the slightest movement.

It appears that for several years he has been nearly deaf with the right ear, and for the last six weeks there has been a discharge, and pain from the mastoid process to the occiput, and down the neck. He has slight cough. At the base of the right lung there is dulness on percussion, bronchial respiration, and a well-marked pleuritic rub. There is much complaint of pain in the head, considerable oppression; an unwillingness to be disturbed, but no delirium or incoherence.

Radatur caput,
Embrocatio communis,
Inf. Serpentariæ,
Ammon. Carb., gr. iv.
Pulv. Cretæ comp., gr. xv,
6tis horis.
Ung. Hydrarg. Fort. axillis infricandum.

July 24th.—Dulness at the base of the left lung. Condition of the right lung and other symptoms much the same.

Repetatur mistura,
Capiat pilulam, Antimonii Opiatam,
c. Hydrarg. Chloridi, gr. j, ter in die.
Empl. Lyttæ lateri dextro.

25th.—Cough, and rust-coloured expectoration; less febrile disturbance.

Repetatur pilula omni nocte tantum.
Julepum Acidi Nitrici c.
Vini Ipecacuanhæ, ♏x, 6tis horis.

27th.—Profuse partial sweats. The pleuritic rub on the right side audible higher up; signs of consolidation remain. On the left side moist râles, audible at the base; expectoration tenacious and puriform.

Inf. Serpentariæ, Jul. Ammon. Acetat., āā ℥ss, c.
Ammon. Carb., gr. iv, 4tis horis.

28th.—Diarrhœa; dulness on the right side more extensive; skin hot and dry; there has been no delirium since admission.

Enema Amyli, c.
Tinct. Opii, ♏xx, statim.
Repetatur mistura.

30th.—He appears to be sinking; quite sensible. At 4.15 p.m. he died. About an hour before death he fell into a convulsion, with contracted pupils, which continued to the end.

Examination fourteen hours after death.—Behind right ear, under the integuments, extending down to the occipital condyle, tissue sloughing. The lining of the meatus auditorius externus thickened and vascular; the membrana tympani in the same condition; no ulceration. The tympanum full of dark-coloured exudation of the consistence of putty. The roof and posterior wall carious. The part of the occipital bone forming the floor of the lateral sinus carious, and the dura mater upon it sloughing. The cavity of the sinus and the internal jugular vein contain an adhesive clot, which in the centre is soft and puriform. Surface of arachnoid slightly greasy; pia mater hyperæmic; convolutions rather flattened. Lateral ventricles enlarged, their lining membrane faintly granular. Section of white substance of brain mottled, and presenting numerous vascular points. (Death by asphyxia.)

In the right pleura twenty ounces of opaque serum with fibrinous flakes. Left lung partially adherent; at the base purulent effusion to the extent of several ounces. In the subjacent pulmonary tissue several lobules in a state of suppuration and disintegration. The upper lobe partially solidified and œdematous; in the right lung also, many patches of suppurating lobular pneumonia. In the pericardium an ounce and a half of clear serum; heart normal, except slight thickening of the aortic valves; abdominal viscera healthy; no trace of tubercle in any organ.

The anatomical relations of the ear to the veins would lead us, prior to experience, *to anticipate that affection of the chest would not be an uncommon result of necrosis of the mastoid cells;* and these, with other recorded observations, show that the lungs, as well as the brain, must be regarded as obnoxious to secondary affections from chronic disease of the ear.

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